CERTIFICATE

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This is to certify that the thesis entitled "**Analysis of Sensorineural Hearing Loss in Chronic Suppurative Otitis Media"** is the bonafide work of **Dr. Naveen Zachariah Philip Mathew** done under the guidance and supervision of **Dr. George Zacharias, MS, DLO** in the Department of E.N.T, PSG Institute of Medical Sciences and Research, Coimbatore in fulfillment of the regulations of Dr. MGR Medical University for the award of M.S. Degree in Oto-Rhino-Laryngology.

DR. GEORGE ZACHARIAS

PROFESSOR & HOD

DEPARTMENT OF E.N.T

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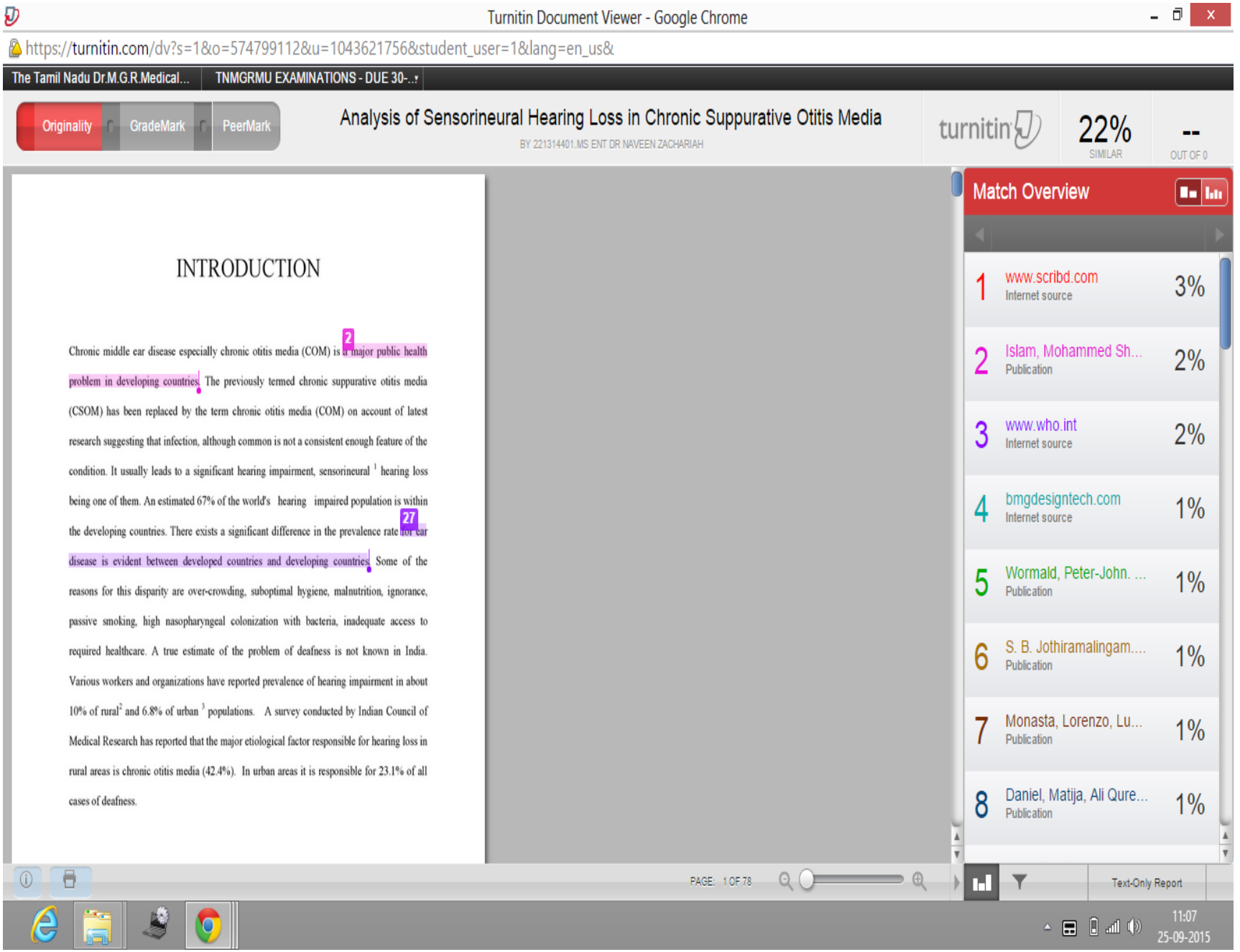
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INTRODUCTION
Chronic middle ear disease especially chronic otitis media (COM) is a major public health problem in developing countries. The previously termed chronic suppurative otitis media (CSOM) has been replaced by the term chronic otitis media (COM) on account of latest research suggesting that infection, although common is not a consistent enough feature of the condition. It usually leads to a significant hearing impairment, sensorineural [1] hearing loss being one of them. An estimated 67% of the world's hearing impaired population is within the developing countries. There exists a significant difference in the prevalence rate for ear disease is evident between developed countries and developing countries. Some of the reasons for this disparity are over-crowding, suboptimal hygiene, malnutrition, ignorance, passive smoking, high nasopharyngeal colonization with bacteria, inadequate access to required healthcare. A true estimate of the problem of deafness is not known in India. Various workers and organizations have reported prevalence of hearing impairment in about 10% of rural[2] and 6.8% of urban [3] populations. A survey conducted by Indian Council of Medical Research has reported that the major etiological factor responsible for hearing loss in rural areas is chronic otitis media (42.4%). In urban areas it is responsible for 23.1% of all cases of deafness.
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INTRODUCTION

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Chronic otitis media is a chronic infection of middle ear cleft characterized by tympanic membrane perforation and inflamed middle ear lining mucosa. It is well known entity prevalent worldwide.

PSG Institute of Medical Sciences & Research
Institutional Human Ethics Committee

Recognized by The Strategic Initiative for Developing Capacity in Ethical Review (SIDCER)
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July 7, 2014

To
Dr Naveen Zachariah Philip Mathew
Postgraduate
Department of E N T
PSG IMS & R
Coimbatore

The Institutional Human Ethics Committee, PSG IMS & R, Coimbatore -4, has reviewed your proposal on 13th June, 2014 in its expedited review meeting held at IHEC Secretariat, PSG IMS&R, between 10.00 am and 11.00 am, and discussed your study proposal entitled:

"Analysis of sensorineural hearing loss in chronic suppurative otitis media"

The following documents were received for review:

1. Duly filled application form
2. Proposal
3. Informed consent forms
4. Assent form
5. Parental consent form
6. Proforma
7. CV
8. Budget

After due consideration, the Committee has decided to approve the study.

The members who attended the meeting at which your study proposal was discussed are as follows:

Name	Qualification	Responsibility in IHEC	Gender	Affiliation to the Institution Yes/No	Present at the meeting Yes/No
Dr P Sathyan	DO, DNB	Clinician, Chairperson	Male	No	Yes
Dr S Bhuvaneshwari	M.D	Clinical Pharmacologist Member - Secretary	Female	Yes	Yes
Dr Sudha Ramalingam	M.D	Epidemiologist Alt. Member - Secretary	Female	Yes	Yes
Dr Y S Sivan	Ph D	Member –Social Scientist	Male	Yes	Yes
Dr D Vijaya	Ph D	Member – Basic Scientist	Female	Yes	Yes

The approval is valid for one year.

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We request you to intimate the date of initiation of the study to IHEC, PSG IMS&R and also, after completion of the project, please submit completion report to IHEC.

This Ethics Committee is organized and operates according to Good Clinical Practice and Schedule Y requirements.

Non-adherence to the Standard Operating Procedures (SOP) of the Institutional Human Ethics Committee (IHEC) and national and international ethical guidelines shall result in withdrawal of approval (suspension or termination of the study). SOP will be revised from time to time and revisions are applicable prospectively to ongoing studies approved prior to such revisions.

Kindly note this approval is subject to ratification in the forthcoming full board review meeting of the IHEC.

Yours truly,

27.6.17

SECRETARY
PSG IMS&R
COIMBATORE-641004
INSTITUTIONAL HUMAN ETHICS COMMITTEE

Dr S Bhuvaneshwari
Member - Secretary
Institutional Human Ethics Committee

DECLARATION

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I hereby declare that this study dissertation entitled "**Analysis of Sensorineural Hearing Loss in Chronic Suppurative Otitis Media"** was prepared by me under the direct guidance and supervision of Professor of E.N.T, Dr. George Zacharias, MS,DLO, PSG Institute of Medical Sciences & Research, Coimbatore.

This dissertation is submitted to the Tamil Nadu Dr. MGR Medical University in fulfillment of the University regulations for the award of M.S. degree in Oto-Rhino-Laryngology. This dissertation has not been submitted for the award of any other Degree or Diploma.

DR. NAVEEN ZACHARIAH PHILIP MATHEW

CERTIFICATE BY THE GUIDE

This is to certify that the thesis entitled "**Analysis of Sensorineural Hearing Loss in Chronic Suppurative Otitis Media"** is the bonafide work of Dr. Naveen Zachariah Philip Mathew done under my direct guidance and supervision in the Department of E.N.T, PSG Institute of Medical Sciences and Research, Coimbatore in fulfillment of the regulations of Dr. MGR Medical University for the award of M.S. degree in Oto-Rhino-Laryngology.

DR. GEORGE ZACHARIAS

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DEPARTMENT OF ENT

ACKNOWLEDGEMENTS

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I would like to extend by heartfelt gratitude to those who have helped me in the completion of this dissertation.

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I take this opportunity to recognize the efforts by E.N.T department staff, **Mrs. Chitrapriya, Mrs. Gomathi, Mrs. Anandhi, Mrs Parimala, Mrs Hema Pavithra** for helping me collect all necessary patient details. I also want to mention a special thanks to the audiology technicians **Mrs. Prema and Mr. Keerthiraj** for their ever present support in the audiology session.

I am grateful for the love and support of my parents and my brother who have been completely instrumental in my ability to put forth this dissertation. I am deeply indebted to my colleagues, seniors and juniors who have really helped in the completion of the study.

I would like to express my sincerest thanks to **Dr. S. Ramalingam**, Principal and the Ethics Committee, PSG IMS & R for having consented to this study.

I sincerely thank **Dr. Vimal Kumar Govindan**, Medical Director, PSG IMS & R for sanctioning the required hospital resources and personnel needed for performing this study.

Finally my heartfelt appreciation & greatest thanks to all the **patients**. Their invaluable time and patience has been primordial to the completion of this study.

INTRODUCTION

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Chronic middle ear disease especially chronic otitis media (COM) is a major public health problem in developing countries. The previously termed chronic suppurative otitis media (CSOM) has been replaced by the term chronic otitis media (COM) on account of latest research suggesting that infection, although common is not a consistent enough feature of the condition. It usually leads to a significant hearing impairment, sensorineural [1] hearing loss being one of them. An estimated 67% of the world's hearing impaired population is within the developing countries. There exists a significant difference in the prevalence rate for ear disease is evident between developed countries and developing countries. Some of the reasons for this disparity are over-crowding, suboptimal hygiene, malnutrition, ignorance, passive smoking, high nasopharyngeal colonization with bacteria, inadequate access to required healthcare. A true estimate of the problem of deafness is not known in India. Various workers and organizations have reported prevalence of hearing impairment in about 10% of rural[2] and 6.8% of urban [3] populations. A survey conducted by Indian Council of Medical Research has reported that the major etiological factor responsible for hearing loss in rural areas is chronic otitis media (42.4%). In urban areas it is responsible for 23.1% of all cases of deafness.

Chronic otitis media is a chronic infection of middle ear cleft characterized by tympanic membrane perforation and inflamed middle ear lining mucosa. It is well known entity prevalent worldwide.

Various studies show prevalence of chronic otitis media in India to range from 2 to 15 %.In a study by Bluestone [4], India was considered to have a low prevalence area of 2% while a study from Haryana by Verma [5] reported it as high as 15.3% .

The round window membrane has been analyzed for it contribution to sensorineural hearing loss in chronic otitis media.[6 -12]. Round window membrane is a semipermeable membrane, which is breached by toxins released during the course of COM and causes biochemical changes in perilymph and endolymph finally leading to destruction of Organ of Corti [10].It has been observed that chronic inflammation enhances increased vascular and macromolecular (protein) permeability within the perilymphatic space.[13]

Different investigators have different views and opinions over the relation between the duration of disease, type of pathology and development of sensorineural hearing loss in COM. Positive co-relation between duration and type of disease and development of sensorineural hearing loss implies that earlier the attempts are made to eradicate the disease, much better would be the outcome after treatment. A definitive co-relation between the duration or type of disease and development of sensorineural hearing loss would mandate the need for early detection and treatment of Chronic Otitis Media as a means to prevent sensorineural hearing loss.

Hence the present study is being undertaken to analyze clinically with respect to age of patient, duration of disease and the type of COM on the development of sensorineural hearing loss.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Global burden of Chronic Otitis Media and regional prevalence-

Chronic otitis media (COM) is a significant cause of hearing loss, especially in the developing world[14]. As the name suggests, it is a long standing disease with a host of complications and sequelae; each of which can have permanent, long standing effects on communication, language development, cognitive and psychosocial development, educational performance and achievement, particularly in children[15].

Prevalence studies of COM show considerable inter-study variation. This is probably because of variations in definition, statistical sampling methods, underreporting of cases and large lacunae in available data from a number of geographical areas and age groups[16,15]

Prevalence surveys done by the WHO, report the overall global burden of disease from COM to be about 65-330 million people, 60% of who suffer from significant hearing impairment[15.]In addition, COM accounts for about 28, 000 deaths, mainly attributable to brain abscess and meningitis. [15,16]

Over 90% of the cases of COM are seen in the countries of the South-east Asia and Western Pacific regions, Africa and regions of the Pacific Rim. It is uncommon in the Americas, Europe, the Middle East and Australia.[15.] Though there are reports of a high prevalence of COM in Aboriginal-Australian children.[17] A survey of ear, nose and throat disorders done in Rural India, shows a 4.31% prevalence of ENT disorders, 36.6% of whom had ear problems; the most common diagnosis among these patients being COM.[18]

As regards the age distribution of COM, it is predominantly a disease of childhood. [19,20,21,22] Studies from a tertiary hospital in Kolkata show that 31.2% of COM cases were from the pediatric age group. Most of the patients with COM fall into the 0-10 year bracket[19].Probably as a result of this, COM studies have been predominantly conducted on a pediatric population. In South India, the prevalence of COM among children was found to be 6%.[24]A similar figure (5%) is reported among children in Nepal.[24]

At the other end of the age spectrum, COM seems also to be the most common ENT disease among the geriatric population. Okoye et al, in their study on otolaryngological disease in the geriatric population (above 60 years) reported a 55.2% prevalence of ear disease, COM being the most common diagnosis.[25]

The direct and indirect costs related to the disease, drugs and their adverse effects and the hearing impairment constitutes a significant burden to the individual and the society. In a study done in Nigeria the cost of treating COM per patient per year was more than the national monthly minimum wage.[27] To the best of our knowledge, there have been no studies on the Epidemiology, clinical profile or hearing loss patterns caused by COM, from Southern India on the population group between 18-60 years an age group that forms the bulk of the economically productive group of any society.

ANATOMICAL CONSIDERATIONS:

The ear is composed of the external, the middle and the inner ear. During the 6th week of embryonic life, the 6 hillocks of His appear near the 1st branchial cleft. These tubercles gradually coincide to become auricle. The adult form is attained by the 20th week.

The external auditory canal arises from the 1st branchial cleft. By the 4th month of gestation, the cells multiply from below ectodermal cleft and forms the meatal plug. It is this meatal plug that undergoes remodeling to result in formation of bony meatus. Recanalization begins just lateral to the ear drum and progresses laterally. The external ear canal is complete by the 7th month

The ear drum arises from the 3 germinal layers i.e ectoderm, mesoderm and endoderm, each giving rise to the lateral epithelial layer, the middle fibrous layer and the medial mucosal layer respectively.

The middle ear cleft i.e the eustachian tube, tympanic cavity, attic, antrum and mastoid air cells forms from the endoderm in the tubotympanic recess. This is found arising from the first and second pharyngeal pouch partially. The malleus and incus develops from mesoderm of the 1st arch whereas stapes develops from the 2nd arch except the footplate and the annular ligament which arises from the otic capsule.

The inner ear is the first of the special senses to develop in man. The evolution of the inner ear begins around the 21st day of embryonic life and is completely from the 4th month. The ectoderm in this region of the rhombencephalon thickens into the auditory placode which invaginates to

become the otocyst. This goes on to form the endolymphatic sac, its duct, the semicircular ducts, saccule, the utricle and the cochlea. The cochlea is completely developed by 5^{th} month of gestation.

The external ear extends from the auricle laterally till the tympanic membrane medially. The entire pinna, apart from the lobule, and the outer part of external acoustic meatus are derived from a single framework of yellow elastic cartilage which is covered by skin. This is attached tightly to the perichondrium over the lateral surface and it is loosely attached over the medial surface. The external auditory meatus extends from the concha till the tympanic membrane. It is 24 mm in length along the posterior wall. There are two parts of the canal namely the cartilaginous and the bony part. The cartilaginous part measures about 8 mm and is the outer part of the canal. The skin, covering this region is thicker and it contains ceruminous glands and pilosebaceous glands. The hair follicles are limited only in the external canal. The bony part forms inner two-thirds and measures 16 mm. Skin lining this region is thinner and in continuity with the tympanic membrane. It is free of hair follicles and cerumen containing glands.

The tympanic membrane forms the medial limit of the external ear.it is oriented obliquely, measuring about 9-10mm in height. It is divided into two parts, the pars tensa and the pars flaccida. Pars tensa forms most of the tympanic membrane. The central part is withdrawn inwards near the tip of the malleus and this region is called Umbo. Bright cone of light is usually found radiating from the tip of malleus extending till the periphery of the anteroinferior quadrant. Pars flaccida also known as Sharpnell's membrane is situated above the lateral process of malleus between the notch of Rivinus and the anterior and posterior malleal folds. It appears pink and not flaccid. The tympanic membrane is divided into 3 layers (i) The epithelial layer is outer most, found in

continuity with the meatus skin lining (ii) The mucosal layer is innermost, continuous from the mucosa in the middle ear. (iii) Fibrous layer is in middle, encloses the handle of malleus. This layer has 3 types of fibers, namely the radial, circular and parabolic fibers. The middle ear is divided into the epitympanum, mesotympanum and hypotympanum.The tympanic cavity consists of:

- 3 Compartments
- 6 walls
- 3 Ossicles
- 2 muscles
- Chorda tympani
- Tympanic plexus
- Eustachian tube
- Mastoid air cell system

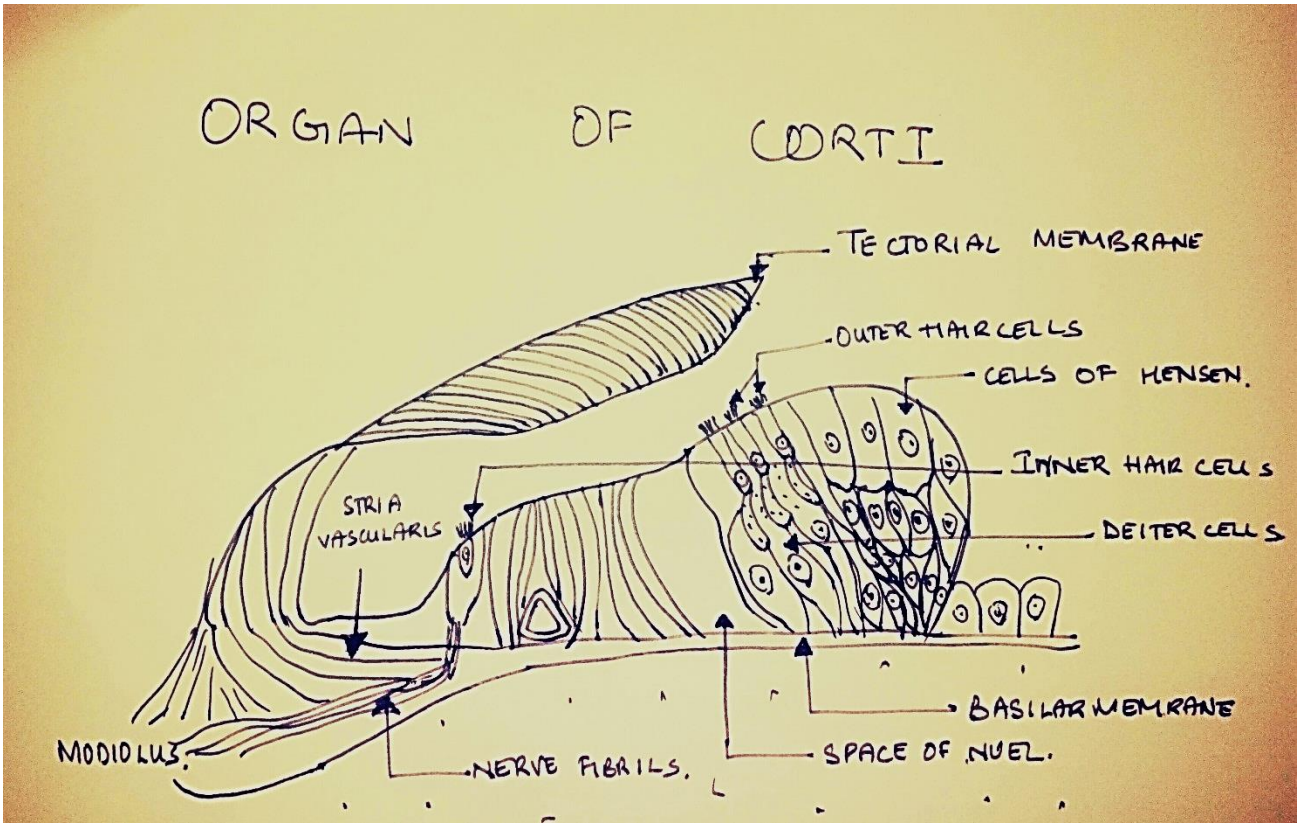
The middle ear cavity has a medial wall (promontory, facial canal, round and oval windows, processus cochleariformis, ponticulus, lateral wall (tympanic membrane, scutum, etc), posterior wall (aditus, mastoid air cells, pyramid, etc). Its anterior wall has the Eustachian tube opening and the tensor tympani. The caroticotympanic nerves also enter the cavity through the anterior wall. The chorda tympani enters the cavity from its posterior wall (after arising from the vertical segment of the facial nerve).It ascends and travels anteriorly between the handle of the malleus and the incus, just below the attachment of the tensor tympani muscle and exits the cavity through a foramen adjacent to the canal of Hugier.

The 3 middle ear ossicles are the hammer (malleus), the anvil (incus) and the stirrups (stapes). Malleus has a head, handle, neck, a lateral process and anterior process. The head and neck of malleus lie in the epitympanum. The lateral process forms a knob-like projection and gives attachment to anterior and posterior malleal folds. The incus consists of the body and short process both of which lie in the epitympanum. The long process is attached to the head of stapes. The

stapes is divided into the head, foot plate, neck, anterior and posterior crura. It is attaches to the oval window by the annular ligament. The ossicles serve as an energy transducer, by transmitting sound energy from the tympanic membrane across the oval window into the fluid filled labyrinth.

The inner ear also called the labyrinth is an organ of hearing and balance. It contains the bony and membranous labyrinth. Membranous labyrinth contains 2 fluids, the endolymph (which is in the scala media of the cochlea) and perilymph is seen in the space separating the membranous and bony labyrinth, scala tympani and vestibuli). The bony labyrinth has three parts: the vestibule, the semicircular canals and the cochlea. The membranous labyrinth consists of the cochlear duct, the utricle, saccule, the endolymphatic duct and sac and three semicircular ducts

The organ of Corti is situated on the basilar membrane. It is the sense organ of hearing and balance. The organ of Corti contains the tunnel of Corti, hair cells, supporting cells, tectorial membrane. The inner and outer rods form the tunnel of Corti. The hair cells undergo depolarization depending on the tectorial membrane movement pattern at a given frequency. The outer and inner hair cells are arranged in a single and 3 rows respectively. The inner hair cells are richly supplied by afferent cochlear fibres and it is probably more important in transmission of auditory impulses. The outer hair cells mostly receive efferent innervations from the Olivary complex. It is concerned with modulating the function of the inner hair cells. There are a total of 3500 inner hair cells and they are flask shaped. The outer hair cells are 12000 in number and they are cylindrical.



Organ of Corti

Acoustics:

Sound is an oscillation in pressure, stress particle displacement and velocity propagated through an elastic or viscous medium. To further simplify, a sound wave occurs as a result of compression and rarefaction of molecules of the medium through which it is travelling. The sound velocity differs from media to media based on their density.

Frequency is total number of oscillations achieved in one second. The unit of frequency is Hertz (Hz) named after the german scientist Heinrich Rudolf Hertz. A sound of 1000 Hz means 1000 cycles occurring in a second.

Pure tone is a single frequency of sound. In single pitch audiometry the threshold of hearing is measured in decibels for varied pure tones ranging from 125 to 8000 Hz.

Complex sound is a sound which contains many frequency. Human voice is an example of complex sound.

Pitch is a subjective perception of the frequency of sound. Frequency and pitch are directly proportional to each other.

A complex sound consists of basic frequency, i.e. the lowest frequency in which a source is set into vibrations. Overtones represent the waves of higher frequency contributing to the net sound. Overtones determine the timber of sound.

Intensity is the sound strength which determines its loud nature. It is usually calculated in decibels. At an approximate distance of 1 metre, the intensity of

Whisper = 30 dB

Normal conversation = 60 dB

Shout = 90 dB

Discomfort = 120 dB

Pain = 130 dB

Loudness is a subjective term for intensity of sound. It is often referred to as the amplitude and is measured in decibels.

Decibel (dB) is the logarithmic ratio among two sounds which is the sound being detailed and sound of reference. It is denoted as 1/10th of a bel. It is named after Sir Alexander Graham Bell. Sound can be measured as power. Level of sound pressure is the measure of sound in audiology. SPL is compared in reference of sound which contained an SPL of 0.0002 dynes/cm2 or 20μ Pa (micropascals) this approximately corresponds to the threshold in normal subjects with hearing under normal limits at 1000 Hz. Decibel is used to avoid large figures of sound pressure level.

$$\text{Sound in dB} = 10 \log \frac{\text{Power of S1}}{\text{Power of S0}}$$

Noise is described as an aperiodic complex of sound. The three varieties of noise are:

(a) White noise: this consists of all the frequencies in the entire audible spectrum. It can be compared to white light which contains all the colours in the visible spectrum. This is a broad-band noise generally used for purpose of masking.

(b) Narrow band noise : This is a form of white noise that contains specific frequencies which are both higher and lower than the sound to be filtered out. Thus its frequency range is smaller than that of broad-band white noise. This is also used in masking specific test frequency during pure tone audiometry.

(c) Speech noise: All noises occurring within the speech range of frequency i.e 300-3000 Hz are called speech noise.

Masking is the phenomenon of inducing inaudibility of a sound by producing another synchronously. Clinical audiometry exploits this method for several tests. Masking is achieved when one ear is occupied by a sound while the other ear is being tested. Masking of ear which is not tested is very essential in all forms of bone conduction tests, while in case of air conduction tests, it is required only when the relative difference in hearing between two ears exceeds more than 40 decibels.

In clinical audiometry, one ear is occupied by a sound while the other ear is being tested. Masking of ear which is not tested is very essential in all forms of bone conduction tests, while in case of air conduction tests, it is mandated only when the relative difference in hearing is more than 40 decibels.

Wide range of human voices falls in this range. PTA is the average threshold measure of all three frequencies of hearing. This roughly equal to the speech reception threshold.

Hearing level is the sound pressure level which is delivered by an audiometer at a respective frequency. It is measured in decibels. The reference is maintained at audiometric zero. If an audiometer produces a sound at 70dB, it is represented as 70dB HL.

Physiology of hearing:

Sound waves travel through external auditory meatus and produce vibrations in the tympanic membrane. Vibrations from tympanic membrane travel through malleus and incus and reach the stapes resulting in the movement of stapes. Movements of stapes produce vibrations in the fluid of cochlea. These vibrations stimulate the hair cells in the organ of Corti. This in turn, causes generation of action potential in the auditory nerve fibers. When auditory impulses reach the cerebral cortex, the perception of hearing occurs. Thus, during the process of hearing, ear converts energy of sound waves into action potentials in the auditory nerve fibers. This process is called sound transduction.

Role of inner ear:

Traveling wave: Movement of foot plate of stirrup against oval window causes movement of perilymph in scala vestibuli. This fluid does not move all the way from oval window to round window through helicotrema. It immediately hits the vestibular membrane near oval window. This causes movement of fluid in scala media, since the vestibular membrane is flexible. This causes bulging of the basilar membrane towards scala tympani. This increases the elastic tension in basilar fibres in the portion of the basilar membrane. This tension initiates a wave, which travels along basilar membrane towards the helicotrema.

Resonance point: It is a part of basilar membrane, which is activated by traveling wave. Initially each wave is weak. When it travels through the basilar membrane from base towards apex, the wave becomes stronger and at one point it becomes very strong and activates the basilar membrane. This resonance point of the basilar membrane immediately vibrates back and forth. The traveling

wave stops here. Distance between stapes and resonance point is inversely proportional to frequency of sound waves reaching the ear. Wave generated by high pitched sound disappears near the base of the cochlea, medium-pitched sound reaches half of the way and wave generated by low pitched sounds travel the entire distance of the basilar membrane.

Excitation of hair cells:

Stereocilia of hair cells in organ of corti are embedded in tectorial membrane. Hair cells are tightly fixed by cuticular lamina reticularis and the pillar cells. When travelling wave causes vibration of basilar membrane at the resonance point, the basilar fiber, pillar cells, hair cells and lamina reticularis move as a single unit. It causes movements of stereocilia leading to excitement of hair cells and generation of receptor potential.

Sound Transduction:

It is type of sensory transduction in the hair cells in the organ of Corti by which sound energy is converted into action potentials in the auditory nerve fiber. Three types of electrical events that occur during sound transduction are:

(1) Receptor potential or cochlear microphonic potential

(2) Endocochlear potential or endolymphatic potential

(3) Action potential in auditory nerve fiber

Role of hair cells:

Inner hair cells and outer hair cells have different roles during sound transduction. The inner hair cells are responsible for sound transduction, i.e. these receptor cells are the primary sensory cells, which causes the generation of action potential in auditory nerve fibers. Outer hair cells have a different action. These hair cells are shortened during depolarization and lengthened during hyperpolarisation. This process is called electromotility. This action of outer hair cells facilitates the movement of basilar membrane and increases the amplitude and the sharpness of sound. Hence, the outer hair cells are collectively called cochlear amplifier.

Role of efferent nerve fibers of hair cells:

They play an important role during sound transduction by releasing acetylcholine. Efferent nerve fiber to inner hair cell terminates on the auditory (afferent) nerve fiber where it leaves the inner hair cell. It controls the generation of action potential in auditory nerve fibre by inhibiting the release of glutamate from inner hair cells.

Theories of hearing:

(1) Telephone theory: It was postulated by Sir Rutherford in 1880. It is also called frequency theory. According to this theory, the cochlea plays a simple role of a telephone transmitter. In telephone, sound vibrations are converted into electrical impulses, which are transmitted by cables to the receiving end. Where electrical impulses are converted to sound waves. Similarly, cochlea converts sound waves into electrical impulses of same frequency.

Impulses are transmitted by auditory nerve fibres to cerebral cortex, where perception and analysis of sound are done. It is approximated that, the nerve fibres can transmit maximum of thousand impulses per second. Thus, the telephone theory fails to explain the transmission of sound waves with frequency above 1000 cycles per second.

(2) Volley theory: Wever postulated this theory in 1949. According to this theory, the impulses of sound waves with frequency above 1000 cycles per second are transmitted by different group of nerve fibers. However there was no evidence to prove it. Thus not accepted by many.

(3) Resonance theory of Helmholtz: This theory was proposed by Helmholtz in 1863. According to this theory analysis of sound frequency is the function of cochlea. Helmholtz named the basilar fibers as resonators and compared them with resonators of piano. When a string in piano is struck, sound with a particular note is produced. Similarly, when the sound with particular frequency is applied the basilar fibres in a particular portion of the basilar membrane are stimulated.

(4) Traveling wave theory: This theory was derived from place theory. It explains the travelling wave generation in basilar membrane.

(5) Place theory: According to this theory, nerve fibers from different portions of organ of corti on basilar membrane give response to sounds of different frequency. Accordingly, corresponding nerve fiber from organ of Corti send information to the brain regarding the portion of organ of corti that is stimulated. Many evidences are present to support the place theory. E.g. If a person is

exposed to loud noise of a particular frequency for a long period, he becomes deaf for that frequency. It is found that the specific portion of organ of Corti is destroyed.

Auditory Pathway:

Hair cells in the organ of Corti are the receptors of auditory sensation. All hair cells are innervated by afferent and efferent nerve fibers. The afferent cells forms the auditory pathway. First order neurons of auditory pathway are the bipolar cells of spiral ganglion. Their long processes leave the ear as cochlear nerve fibers and enter medulla oblongata, where they divide into dorsal and ventral cochlear nucleus on same side of medulla.

These act as second order neurons, where they run as different groups to cross the superior olivary nucleus, lateral lemniscus and recticular formation. The third order neurons are in the superior olivary nuclei and nucleus of lateral lemniscus. Fibers from medial geniculate body go the temporal cortex, via the internal capsule as auditory radiation. Some fibers run to inferior colliculus which are responsible for reflex movements of head in response to auditory stimuli. The cortical auditory centers in the temporal lobe of cerebral cortex lie are the primary auditory area[41,42] and Wernicke area. The secondary auditory area is 22.

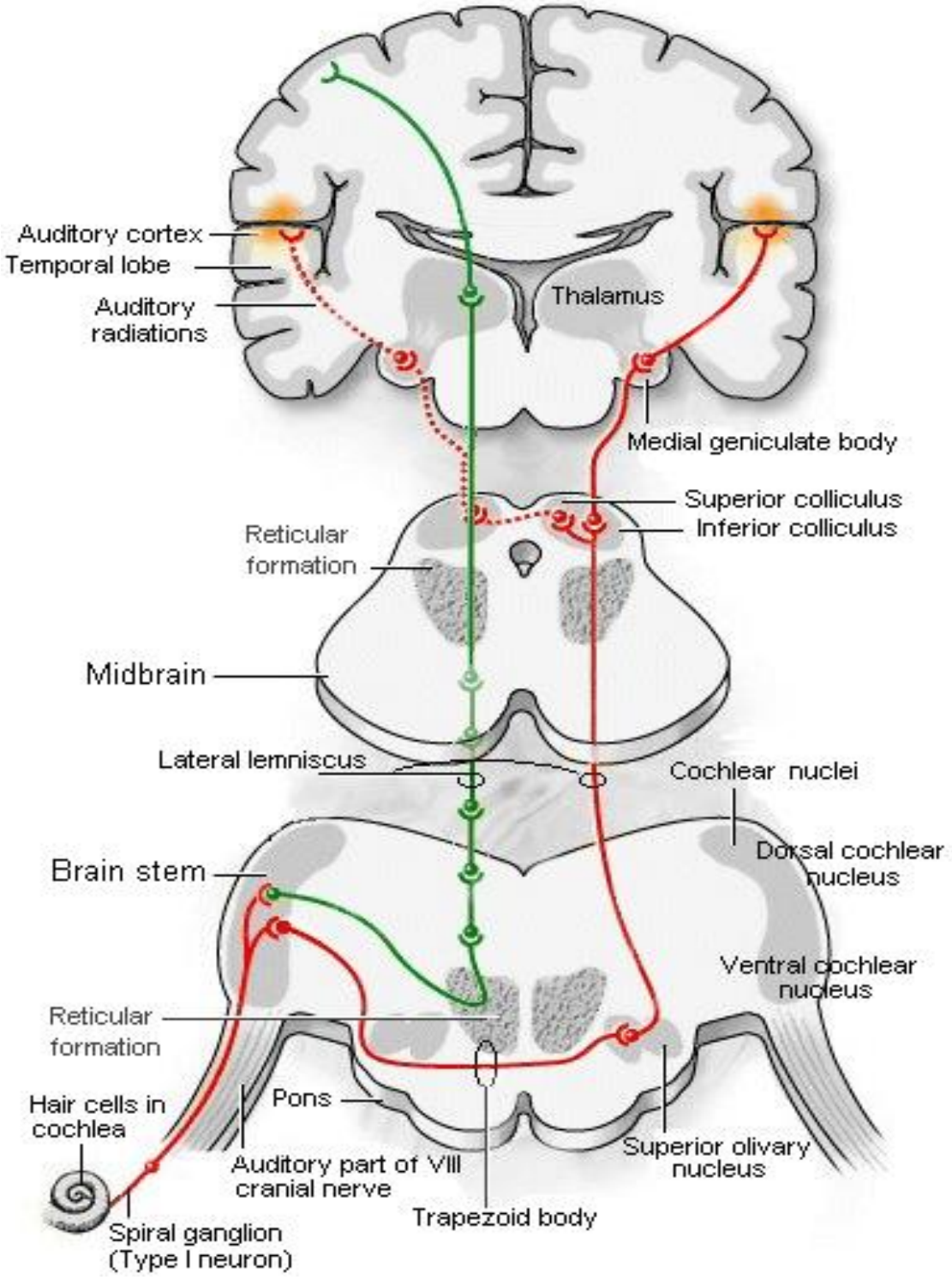


Figure. 2. Auditory Pathway

Hearing loss:

Hearing loss can be of three types (i) Conductive hearing loss (ii) Sensorineural hearing loss (iii) Mixed hearing loss. While auditory function is being assessed, it primordial to decipher the variant and degree of hearing loss; site of lesion and cause of hearing loss. Hearing of an individual can be assessed by clinical examination and audiometric evaluation.

Sensorineural hearing loss: This results from cochlear lesions, VIIIth nerve or central auditory pathways. It may be congenital or acquired. The characteristics of sensorineural hearing loss are:

- A positive Rinne test
- Weber lateralized to better ear
- Bone conduction reduced on Schwabach and absolute bone conduction test.
- Mostly high frequency loss
- No gap between air and bone conduction curve on audiometry
- Loss may exceed 60dB
- Speech discrimination is poor
- There is difficulty in hearing when noise is present

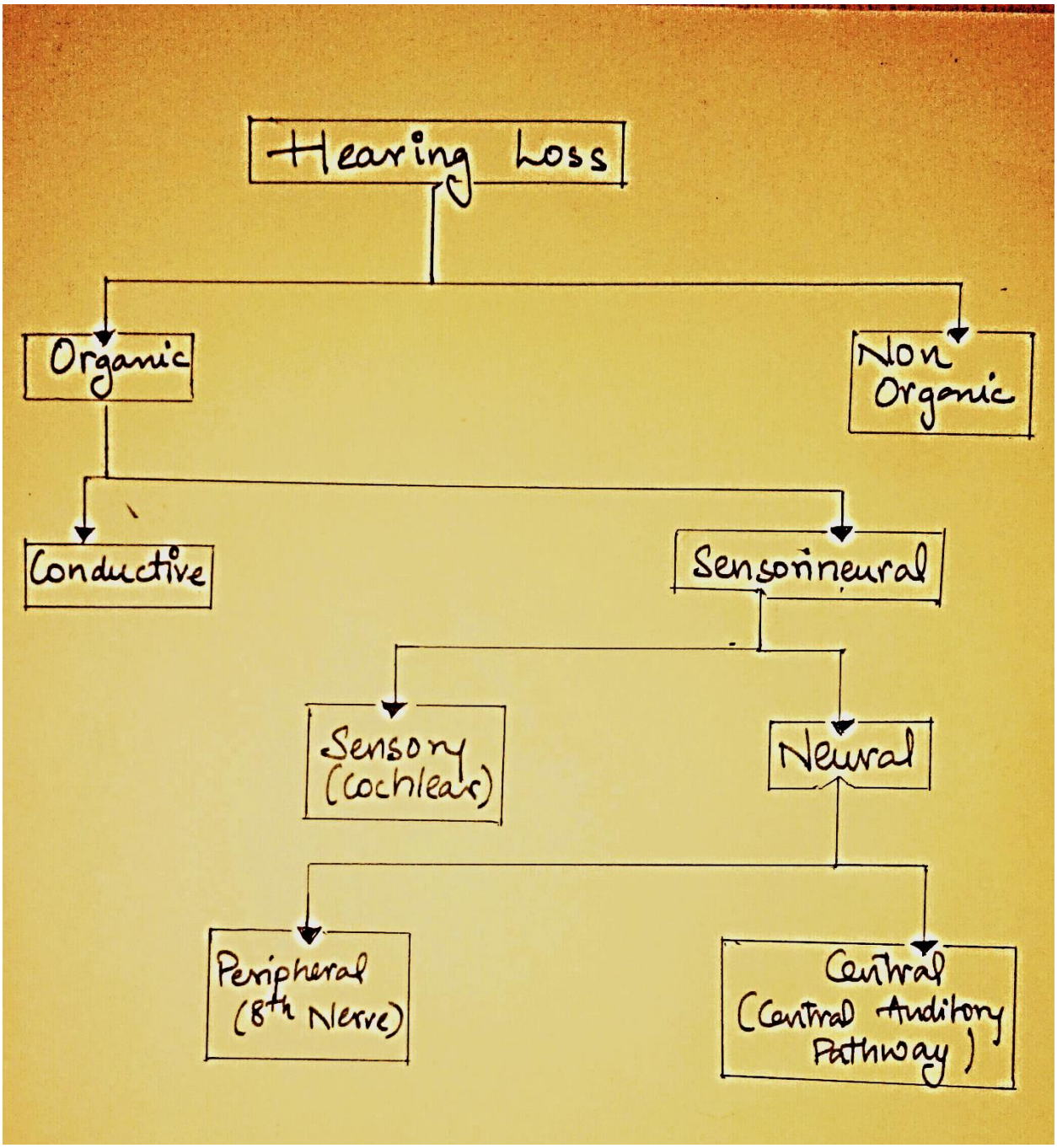


Table: 1: Classification of Hearing Loss

Congenital sensorineural hearing loss is present from birth and is the result of anomalies of the inner ear or damage to the hearing apparatus by prenatal or perinatal factors.

Acquired sensorineural hearing loss appears later in life. The cause may be genetic or non- genetic. The genetic cause of hearing loss may presents late and damages only the hearing. It can even be a component of a larger syndrome with other the body systems being involved. Common causes of acquired sensorineural hearing loss are:

- Infections of labyrinth
- Trauma to labyrinth
- Noise-induced hearing loss
- Ototoxic drugs
- Presbycusis
- Meniere's disease
- Acoustic neuroma
- Sudden hearing loss
- Systemic disorders like diabetes, hypothyroidism, kidney disease and autoimmune disorders.

Specific causes and variants of hearing loss:

A. **Inflammation of the labyrinth**

1. Viral labyrinthitis: viruses usually reach the inner ear by blood stream affecting stria vascularis, endolymph and organ of Corti. Measles, mumps and cytomegalovirus are known to cause labyrinthitis.

2. Bacterial: these infections reach the labyrinth through the middle year or through CSF. sensory neural deafness following meningitis is a known complication .

Bacteria can invade the labyrinth along nerves, vessels, cochlear aqueduct or endolymphatic sac and this may cause complete destruction of the membranous labyrinth.

3. Syphilitic: Sensory neural hearing loss is caused both by congenital and acquired syphilis. Involvement of the inner ear can cause sudden sensorineural hearing loss which may be unilateral or bilateral. Meniere's syndrome with episodic hearing loss, tinnitus and aural fullness. Tullio phenomenon where loud sounds produce vertigo.

B. **Familial Progressive Sensorineural hearing loss**: It is genetic disorder characterized by progressive degeneration of cochlea. It may start late in childhood or in early adult life.

C. **Ototoxicity**: Various chemicals and drugs affect the inner ear and produces sensorineural hearing loss and tinnitus. Symptoms of ototoxicity-hearing loss, tinnitus or giddiness may be noted during treatment or even after completion of entire treatment.

Classification of Ototoxic agents

Aminoglycoside antibiotics:	**Analgesics:**	**Cytotoxic drugs:**
-Streptomycin	- Ibuprofen	- Nitrogen mustard
- Tobramycin	- Salicylates	- Carboplatin
- Gentamicin	- Indomethacin	- Cisplatin
- Neomycin	- Phenyl butazone	
-Amikacin		

Diuretics	**Chemicals**
- Alcohol	
- Furosemide	- Tobacco
- Ethacrynic acid	- Carbon monoxide poisoning

Antimalarials	**Miscellaneous**
-Quinine	
-Chloroquine	- Erythromycin
-Propranolol	- Ampicillin

D. **Noise trauma**:

Hearing loss due to exposure to noise exposure is known in boiler makers, iron smiths, copper smith and artillery men. Now a day's noise trauma has become very significant as it has become an occupational hazard. Hearing loss caused by excessive noise can be divided into two groups:

1. Acoustic trauma: Permanent damage to hearing can occur even on single brief exposure to very intense sound. E.g.: Gunfire; explosion. Sudden loud sounds cause damage to the outer hair cells eventually disrupting the organ of Corti and potential rupture of the Reissner's membrane. In some cases rupture of tympanic membrane and disruption of ossicular chain can happen in severe blast.

2. Noise-induced hearing loss: Here hearing loss occurs following chronic exposure to less intense sounds. This is mostly noted as occupational hazard in people working in noisy environment.

The damage caused by noise trauma depends on many factors like:

a. Frequency of noise

b. Intensity and duration of noise

c. Continuous or interrupted noise

d. Susceptibility of the individual

e. Any pre-existing ear disease.

E. **Sudden Hearing loss**: It is a sensorineural hearing loss that is developed over a period of hours or few days. Hearing loss may be partial or complete. It is mostly unilateral and may be

accompanied by tinnitus or spells of vertigo. Mostly the cause of this sudden deafness remains unclear and termed as idiopathic. The three main etiological factors generally considered are: viral, vascular or rupture of cochlear membrane. The other factors causing sudden hearing loss are:

- Infections
- Trauma
- Vascular
- Ear pathology like Meniere's disease.
- Toxic
- Neoplastic
- Psychogenic

F. **Presbycusis**: Sensorineural hearing loss associated with the aging process in the ear is termed as presbycusis. Generally it manifests at 65 years or even earlier. The four pathological types are:

- Sensory
- Neural
- Strial or metabolic
- Cochlear conductive

The tests for hearing are:

A. Clinical tests of hearing are (i) Finger Friction Test (ii) Watch Test (iii) Speech Tests (iv) Tuning Fork Tests which include (a) Rinne test (b) Weber test (c) Absolute bone conduction test (d) Schwabach's test (e) Bing test (f) Gelle's test.

Rinne tuning fork test: is formulated to find the difference between air conduction with that of bone conduction. In normal conditions, air conduction is more than bone conduction and the tuning fork will be heard loud in the opposite ear canal than when it is placed over the mastoid bone behind.

The alternate method for doing this test is by comparing the sound threshold. In this method the activated tuning fork is held opposite the canal till the patient ceases to hear the sound after which it is placed over the mastoid process. If sound is heard once more, then it is considered that air conduction is worse than the bone conduction thereby suggesting conductive loss and hence Rinne negative. The test is to be done with 256 Hz, 512 Hz and 1024 Hz tuning forks. When done over 3 frequencies, this test also provides an estimate of the air bone gap. False positivity of this test is about 20%.

Weber tuning fork test: This test is used only if asymmetrical or unilateral hearing is seen in patients. The basis of this is by placing a tuning fork in the centre of the head being perceived louder on the ipsilateral side in case of conductive impairment and on the contralateral ear in sensorineural loss.

This difference can be distinctive only, if the examiner has done a clinical hearing test previously and if he knows which ear has better hearing. The test is done by keeping a struck tuning fork on any midline bony prominence, i.e. incisor, nasal bridge. The tuning fork can also be placed over the vertex of the skull in the midline. Then the patient is asked to identify the ear in which the sound is heard or otherwise in which ear the sound is louder. The sound is lateralized to the ipsilateral ear in conductive loss and contralateral ear in case of sensorineural compromise. This test is however not very sensitive nor specific.

Absolute bone conduction test: Bone conduction is measurement of the function of cochlea. Here bone conduction of the patient is compared with the examiner, assuming the examiners have hearing within normal limits. The external auditory canal of both the examiner and the patient are closed, so that ambient noise entering through air conduction route is prevented. In case of conductive hearing loss, the subject and the examiner hears the tuning fork for equal time period. Whereas in case of sensorineural loss, the subject perceives the tuning fork for a brief period of time.

Schwabach's test: Here again the bone conduction of the patient is compared with the examiner, assuming that he has hearing within normal limits. But in this test, the meatus is not occluded. Schwabach's test is reduced in case of sensorineural deafness and lengthened in case of conductive deafness.

The Bing test: it is done on the same basis of Weber test in which closing of the external auditory canal increases the tuning fork sound in the ear to be tested, if conductive hearing loss is present.

It is done by keeping a vibrating tuning fork over the mastoid process and the external auditory meatus is also occluded.

If increase in sound is present then there is less likelihood that there is conductive hearing loss. However, if it remains the same, then it is more likely to be a conductive deafness. The specificity and sensitivity of this test is also very low. Most of the situations normal individuals are identified as conductive deafness. This test is not used widely.

Tuning fork tests should generally be reserved for situations where audiometry is not satisfactory. The results must be interpreted keeping in mind the low sensitivity and specificity.

B. Audiometric tests are (i) Pure tone audiometry (ii) Speech audiometry (iii) Bekesy audiometry (iv)Impedance audiometry.

C. Special tests of hearing are (i) Recruitment (ii) Short increment sensitivity index (SISI) (iii) Threshold tone decay test (iv) Evoked response audiometry (v) Otoacoustic emissions (vi) Central auditory tests (vii) Hearing assessment in children and infants.

Pure tone audiometry: Pure tones are produced by an electronic device called an audiometer. The intensity of these tones can be increased or decreased in steps of 5 dB. Generally thresholds for air conduction are calculated for tones of 125, 250, 500, 1000, 2000, 4000 and 8000 Hz and for thresholds of bone conduction, it is done at 250, 500, 1000, 2000 and 4000 Hz.

The measure of intensity of tones that has to be increased more than the level of normal is considered as the degree of hearing impairment in that particular frequency. Audiogram is a graphical representation of the charted values. The bone conduction threshold is a measurement of function of cochlea. The variations in the air conduction and bone conduction thresholds (A-B

gap) are a measurement of the degree of conductive hearing loss. It is observed that the calibration of audiometer is done in such a way that the perception in a normal person, both the air and bone conduction remains at zero dB and no A-B gap is noted. The turning fork test generally shows AC>BC. When the difference of hearing in the 2 ears is 40 dB or more in thresholds of air conduction then the ear with better hearing is masked so as to not to get a shadow curve in the better ear that is not being tested. In the same way masking is important in all studies of bone conduction. Masking is carried out by delivering a narrow-band noise to the ear that is not being tested. The benefits of pure tone audiogram are (i) It is a measurement threshold for hearing in both air and bone conduction, and also the type and degree of hearing loss. (ii) It is a reference for future record. (iii) Hearing aids can be prescribed only after an audiometery (iv) Speech reception thresholds can be predicted with its help (v) The degree of handicap can be assessed for medico legal issues.

Equipment:

A pure tone audiometer is designed with a set of basic functions. The technical requirements for the instrument are specified by international standard, in which four types are identified based on complexity of functions and working range of various characteristics.

Definition of COM

One of the most important goals of COM research is to achieve a uniform consensus about the definition of COM [8] . According to WHO, COM is defined as a chronic middle ear and mastoid cavity inflammation, presenting with recurrent ear discharges or otorrhoea through a perforated tympanic membrane. The time needed to elapse before labeling the disease as chronic is controversial. In the WHO definition, this is taken as persistent ear discharge for 2 weeks, but most otologists tend to wait for longer periods of time- commonly waiting for as long as 3 months of persistent ear discharge, despite antibiotic therapy, for a diagnosis of COM[2] .A similar definition is stated by the article published by Monasta et al, where COM is defined as a persistent inflammatory process associated with a perforated tympanic membrane draining exudates for more than 6 weeks, which is often associated with cholesteatoma[3]. Whereas, Quereshi et al have diagnosed CSOM as when a permanent tympanic perforation is detected along with middle ear mucositis with or without persistent otorrhoea, with the discharge present for a minimum of 2-6 weeks[7].Therefore, whether otorrhea is needed to define COM is itself a point to consider. However, most studies have used the presence of otorrhoea (either chronic or intermittent), to define the presence of COM.

Acute otitis media (AOM)

Acute otitis media, is usually a short lived inflammation of the middle ear, characterized by rapid onset of one or more signs or symptoms of acute inflammation of the middle ear. These can be earache, tugging at the ear, fever, irritability due to fluid in the middle ear. [28]

The pathogenesis of AOM is multifactorial and involves a host of complex interactions between microbial agents, host immune response, cell biology and anatomy of the middle ear cleft (mastoid, middle ear and Eustachian tube), the nasopharynx, genetic and environmental factors[20,22,29].Viral infections of the upper respiratory tract usually precede or coincide with episodes of AOM[15,20], especially RSV, adenovirus and CMV.[30] These viral infections seem to have a pivotal role in the progression of AOM. As they seem to form an environment conducive for secondary bacterial colonization, adhesion and invasion into the middle ear.[30] These bacteria are believed to enter the middle ear via the Eustachian tube. *S. pneumonia* and *H. influenzae* have been most frequently detected in effusions from cases with recurrent AOM.[31,32,33] The upper respiratory infections in turn lead to congestion of the Eustachian tube and nasopharyngeal mucosa, which prevents normal functioning of the eustachian tube, altering the pressure regulation of the middle ear. In the presence of sustained congestion, nasopharyngeal pathogens gain access to the middle ear, which then go on to further stimulation of inflammation and pus collection, resulting in the clinical symptoms of AOM.[20] This prolonged inflammation can cause the middle ear ossicles to become less mobile and they are subject to resorption.[34]

Recurrent otitis media is defined as three or more episodes of AOM within a 6 month period, or four episodes in one year. [35]

Pathogenesis of COM

The seeds of COM are sown when there is a tympanic membrane perforation in the setting of an acute infection of the middle ear.[2] There a number of suspect factors postulated for the development of COM, however (as is usually the case) none of them are clearly recognized in the literature available. There seems to be a correlation between recurrent AOM and a predisposition to COM.[36] One of the most convincing pieces of evidence for an association between AOM and CSOM, is the decline of the incidence of COM with the widespread use of antibiotics for AOM, suggesting that probably timely and correct management of the acute condition can prevent the development of the chronic form.[37,1] However the liaison between AOM and COM is not an established association and results from a number of studies have produced a number of contradictory results.

On the other hand, Eustachian tube characteristics, genetic, immunological and environmental factors all seem to have definite roles to play in the susceptibility to COM.[38,39,40] More recently, there is interest in the role of a biofilm in the pathogenesis of COM.[1]

The presence of a tympanic membrane perforation, coupled with chronic or inadequately treated middle ear infection allows squamous epithelial migration across the edges of the perforation. Thus the healing of the perforation is affected and it becomes permanent.[7] In developed countries the most common cause of this tympanic membrane perforation is the result of insertion of a ventilation tube.[42,43] In developing countries, tympanic membrane perforation usually occurs as a complication of AOM.[42]

Diagnosis of COM

A history of ear discharge, particularly along with colds, cough or other symptoms of a upper respiratory tract infection should raise the suspicion of COM. Pain is usually not a prominent symptom and is one of the reasons why there is a delay in seeking treatment.[15,20,44,45]

As mentioned earlier, the exact duration of the otorrhoea is controversial; however this information becomes critical only if there is absence of the visibility of the tympanic membrane. In most cases, otoscopy can confirm the diagnosis based on the perforation size, discharge characteristics and the state of the middle ear mucosa.

Based primarily on the otoscopic examination of the tympanic membrane and ear canal, COM can be classified into perforation without evidence of any other disease, tympanic membrane perforation with disease confined to middle ear and tympanum, attic cholesteatoma, disease of the middle ear, epitympanum and mastoid without cholesteatoma, disease involving the middle ear, epitympanum and mastoid with cholesteatoma. [46]

There are other forms of chronic otitis media, which can be thought to comprise a group of non-COM entities. In the current study, we have defined COM by the presence of a persistent, perforated ear drum and a middle ear. In other words, this is could also be known as chronic active mucosal otitis media, chronic oto-mastoiditis and chronic tympanomastoiditis.[2] The forms non-COM chronic middle ear infections include chronic non-suppurative otitis media, chronic otitis media with effusion, chronic secretory otitis media, otitis media with persistent effusion, chronic middle ear catarrh. These all comprise of chronic middle ear fluid collections behind an intact ear drum.[16,47]

Bacteriology of COM

Bacteriologically CSOM cultures usually show a mixed growth. Bacterial isolation rates seem to be higher in adults than in children.[48] The bacteria isolated maybe aerobic *(P. aeruginosa, E. coli, S. aureus, Klebsiella sp*) or anaerobic (Bacteroides, Peptostreptococci)[15,20,29] These organisms are not frequently found in the external canal, but probably proliferate in the presence of trauma, inflammation and the moist environment seen in otitis media.[50] Classically, *P. aeruginosa* was probably the most common organism isolated in pure cultures,[51] and is also the organism most commonly blamed for the deep seated and progressive destruction of the middle ear and mastoid structures by its toxins and various enzymes. Recent studies done by Lee et al have demonstrated the emergence of MSSA also as a significant contributor.[49] The frequency of methicillin resistant *S. aureus* was higher in adults than in children.[49]

Complications of COM

Based on the risk of progression of progression to significant complications, COM can be viewed as mucosal (tubotympanic) or squamous (atticoantral).[52]In comparative studies on these two variants, patients with poorer socioeconomic status were seen in the squamous type. The mucosal type had a tendency for central perforation, whereas the unsafe type had attic and marginal perforations. In the mucosal type, the discharge was mucoid or mucopurulent, whereas unsafe type was often found to have scanty, foul smelling discharge. Complications, as expected were more with the atticoantral variety and included post auricular sinus, subperiosteal abscess, meningitis and brain abscess.[53] Both types can lead to erosion of the ossicular chain, though the risk of erosion is more in the squamous type, due to the presence of cholesteatoma and granulations.[54] Atticoantral

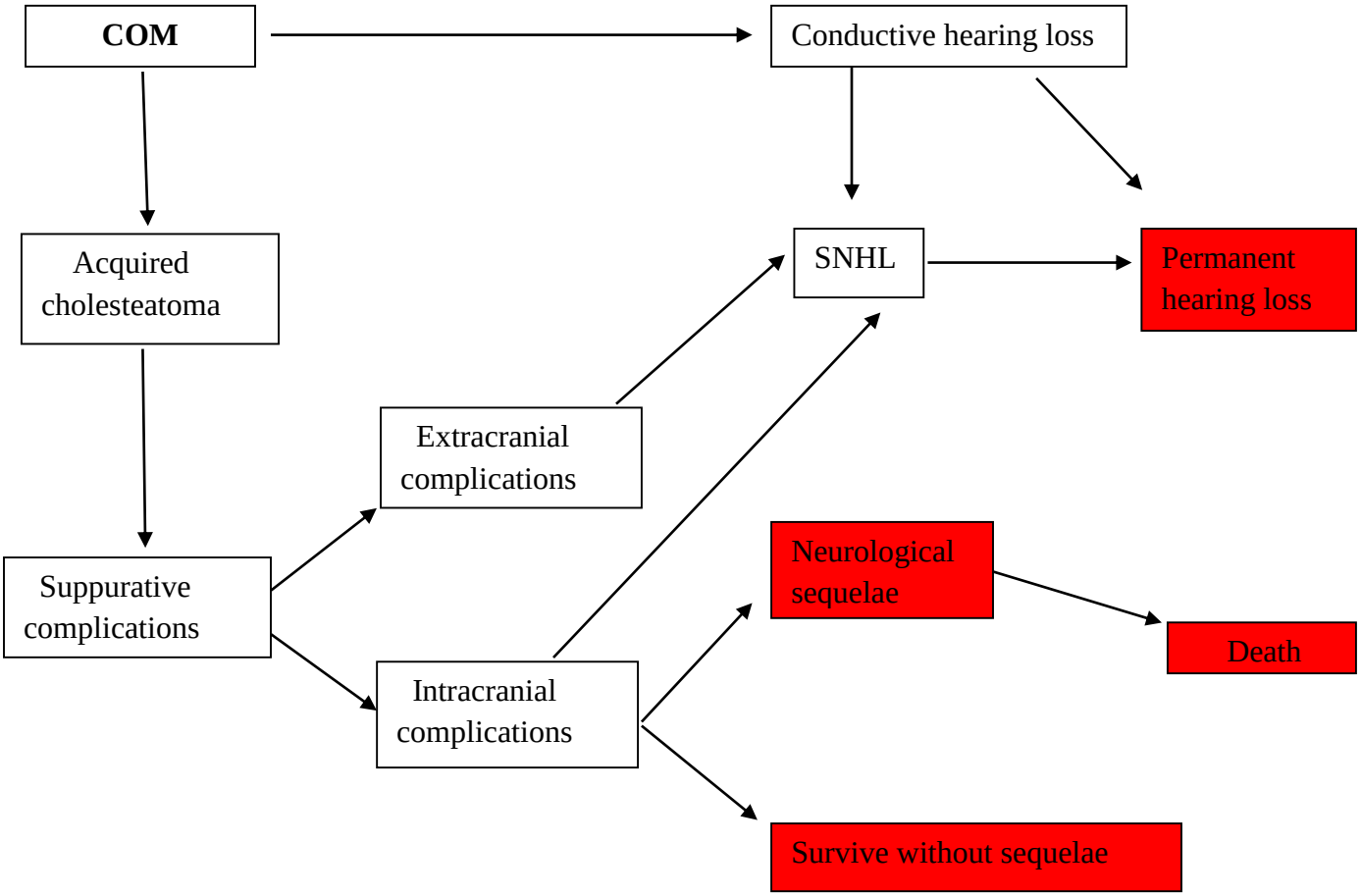
disease can occur in any age group. In a study by Jothiramalingam et al, the most common age group quoted as being 16-25 years. [55]

The cholesteatoma is a destructive disease of the middle ear, commonly seen with atticoantral type of disease[55]. The term cholesteatoma was proposed by Johannes Muller, a German physiologist in 1838. Several theories for the formation of a cholesteatoma have been developed such as the retraction pocket theory, proliferation theory, immigration theory and metaplasia theory. In brief, a retraction pocket seems to be formed due to tubal dysfunction. Cell debris and keratinocytes accumulate in this pocket. The moist keratin is a rich medium for bacterial infection. In the presence of infection, the normal self-cleaning mechanisms are disturbed and thus setting up a vicious cycle of epithelial proliferation, keratinocyte differentiation, maturation, subsequent apoptosis and faulty self-cleansing process. This aggravates the inflammation and stimulating further epithelial proliferation along with the expression of lytic enzymes and cytokines. Bacteria inside the retraction pocket produce antigens which further activate cytokines and lytic enzymes such as ICAM, RANKL,IL-1, IL-2, IL-6, MMP-2 and MMP-9. These go on to activate osteoclast maturation which eventually lead to degradation of extracellular bone matrix, hyperproliferation, bone erosion and thus disease progression.[56,57]

A superadded acute ear infection is characterized by the presence of granulation tissue, foul smelling discharge and ear pain occasionally. Often culture of the ossicles and ossicular suspensory ligaments can yield Pseudomonas and even fungus. [58,59]

Prussack's space is a pouch like space between the pars flaccida of the tympanic membrane and the neck of the malleus. Epitympanic cholesteatomas originate in this shallow pocket. They can break the confines of this pocket in three possible directions. The most common is the posterior route where it penetrates the superior incudal space lateral to the body of the incus. From here it penetrates the aditus ad antrum eventually gaining access to the mastoid.[55,60]

The second most common is the inferior route, through the Pouch of Von Troeltsh, where it descends into the posterior mesotympanum in the region of the stapes, round window, sinus tympani and facial recess. It most commonly involves the facial nerve in its horizontal portion or at the second genu.[55,61] Anterior extension, the third route, is rare.[55] Ossicular erosion seems to be more common among children.[62] Among the ossicles, erosion of the lenticular process of the incus is most common.[63,55] There is some controversy regarding the incidence of erosion of the malleus and the stapes.[55,63,64,65] The complications associated with chronic otitis media can be summarized in the following algorithm.[16]



The red boxes indicate the possible end points of the disease

The extracranial complications include mastoiditis, facial nerve palsy, labyrinthitis and petrous apicitis. The intracranial complications include meningitis, extradural abscess, subdural abscess, brain abscess, sigmoid sinus thrombosis and otic hydrocephalus. The frequency of complications has decreased dramatically with the introduction of antibiotics, more so in developed countries. However lower socio-economic status, poor knowledge and scarce availability of trained ENT surgeons have not caused a similar decrease in developing nations.[66,67,68]

COM and Hearing impairment- global burden and regional prevalence

Hearing impairment is the total or partial inability to hear sound in one or both ears. [69,70] According to WHO, hearing impairment is permanent unaided hearing threshold in the better ear of more than 30 dB in children aged upto 15 years, or more than 40 dB in adults at frequencies of 0.5, 1, 2, and 4 kHz[28]. According to the ASHA, a hearing disorder is the result of impaired auditory sensitivity of the physiological auditory system.[71] In a study conducted at the University Of Wisconsin Medical School by Cruickshanks et al among older adults, hearing loss was defined as pure-tone average of thresholds at 500, 1000, 2000 and 4000 Hz greater than 25 dB in the worse ear. The worse ear is chosen in order to include people with at least one affected ear. The severity of hearing loss can be further classified as mild (>25 and <=40 dB of hearing loss), moderate (>40 and <=60 dB of hearing loss) and marked (>60 dB of hearing loss).[72]

Global estimates of the hearing impairment due to COM are difficult to obtain. According to a report by WHO, COM accounts for 3% to 80% of the burden of hearing impairment.[15] In other words, about 164 million cases of hearing impairment maybe said to be due to COM and 90% of these would be in developing countries.[15]However, this is probably an underestimate and the WHO report states that 200 million seems a more realistic global estimate.[73,74,75] COM probably

contributes more than half the global burden of hearing impairment, eliminating it can reduce the global burden by four-fifths. [15]

In a study conducted at Kenya, hearing loss was documented in 64% of school children with COM and in only 3.4% of children without COM. (76) In a study done on Tanzanian children, 64.7% of COM cases had hearing impairment and 8.8% out of 354 students in a deaf school had hearing impairment due to COM.[77] In India 77% of COM cases had hearing impairment. In a study done on children in Vellore, India, otitis media accounted for 91% of cases of hearing impairment and 53.4% of those with middle ear disease had hearing impairment.[78] The Indian Council for Medical Research has reported that 42.4% of the hearing loss in rural areas is COM and in urban areas, 23.1%.[79]

The HUNT study, a population based cohort study, states that ears with hearing loss after OM (recurrent AOM and COM), in fact age faster than those without. [80]

COM is thus, deservedly, considered to be the most common cause of mild to moderate hearing impairment in children and young people in developing countries. There are many reasons why hearing loss due to COM is more in the developing regions. Apart from the fact that COM and complications due to COM are more common in these regions (as stated in the above paragraphs), the situation is aggravated because of a severe limitation of funding for prevention, early detection and rehabilitation. [68,81]

COM and Conductive hearing loss

Conductive hearing loss (CHL) is a total or partial inability to hear sound in one or both ears because of a mechanical problem in the external or middle ear. That is, the ossicles may fail to conduct sound to the cochlea, or the ear drum may fail to vibrate in response to sound. [70] For the

purpose of diagnosis, a conductive loss is demonstrated by an air-bone gap of 15dB or greater at 500 or 4000 Hz in the ear with the worse hearing.[72]

COM typically produces a mild to moderate conductive hearing loss. [15,82,83,84] The duration of ear discharge increases the degree of hearing loss.[85].This conductive hearing loss results from tympanic membrane rupture and changes in the ossicular chain by fixation or erosion by the chronic inflammatory process. There is a correlation between the site and size of perforation with the degree of hearing loss. With larger, posterior perforations, the degree hearing loss seems to increase.[85,86,87,88] Yung et al found 43 dB HL in a series of big , central perforations.[89] In a study by Ahmad et al it was found that anterior perforations had 18.5 dB of HL versus 29 dB in posterior perforations with 500 Hz. They also reported that the hearing loss is greater with malleolar perforations as compared non-malleolar, unless the perforation involved less than 10% of the tympanic surface membrane.[90]The presence of a cholesteatoma or granulation tissue increases the hearing loss by increasing the degree of ossicular destruction.[83] The hearing loss is more appreciable at lower frequencies as compared to higher frequencies.[91,92,93]

COM and Sensorineural hearing loss

Sensorineural hearing loss (SNHL) is the total or partial inability to hear sound in one or both ears resulting from a dysfunction of the inner ear. It most often occurs when the cilia of the inner ear, the cells that transmit sound from the inner ear, are damaged.[16]

Whether COM can result in SNHL has been a topic of debate for many years. As early as 1955, Gardhengi reported a 44% (22 out of 50 patients) prevalence of SNHL due to COM. [93] Bluvshtien in 1963 reported a 37.5% prevalence.[94] Verhoeven (1961) and Thorburn made observations of cochlear damage in COM. [95] Paparella (1970) and English (1973) reported similar occurrences of

SNHL in patients with COM.[96,97] In 1998 Blakley et al collected audiometric data from 123 patients with unilateral COM and reported that SNHL is associated with COM, with some people being particularly susceptible.[98] Kaur et al studied 100 cases of unilateral COM and reported a 24% incidence of SNHL.[99]

Contrastingly, Dumich et al, after studying 200 consecutive patients, stated that patients with prolonged disease and extensive pathological alterations had increased chances of acquiring SNHL, though not to a level of significance.[100]Similar results were published by Ho et al.[101]

According to most studies, the fact that there is a relationship between COM and SNHL is more or less well established. This has also been demonstrated by objectively analyzing human temporal bone from patients with COM, where pathological changes suggestive of cochlear damage were observed. [102] However, what exactly the relationship is and what are the risk factors for developing SNHL remains controversial.[103]

The possible mechanism by which COM can cause SNHL has been studied by experimental observations using animal models.

The round window seems to playing a crucial part in the process, acting as it were, like a truant gate keeper to the inner ear, allowing the passage of inflammatory substances. The three layer thick round window sits in a in a niche approximately 1 mm in depth and 2 mm in diameter. This niche has no ciliated cells under normal circumstances. Also, mesenchymal remnants in the round window niche and scala tympani are often slow to be resorbed, forming a pocket for pus pooling. These anatomical characteristics of the round window encourage the accumulation, stagnation and absorption of toxic, purulent secretions in to the perilymph.[99,96] Ultimately it results in chemical contamination of the perilymph and damage to the inner ear. Paparella et al, on studying the perilymph of the scala tympani, reported the presence of serofibrinous precipitates and

inflammatory cells in the perilymph. There has also been documented an increase in the LDH levels especially malic dehydrogenase following experimentally inducing middle ear infection in the guinea pig ear. Temporal bone studies and studies of the round window by Paparella et al demonstrate signs of inflammation and increased permeability of the round window and the round window niche. Goycoolea et al in an experimental study on cat ears concluded that all the changes seen were clearly suggestive of an active reaction to inflammation ultimately leading to a change in the permeability of the round window. This permeability change has also been demonstrated by experimentally passing sodium-22 and horseradish peroxidase through the round window and into the labyrinth. [104]

Guo et al in a study in 1994, reported the lipopolysacchariade induced endotoxic damage of the stria vascularis, producing an ion imbalance resulting in changes in the endolymph and dysfunction in the Organ of Corti.[105]

In addition to toxic inflammatory mediators generated by the inflammation of the middle ear, inner ear destruction is also mediated by superadded bacteriological infection. Altered round window permeability to noxious bacterial proteases, has been demonstrated by in vivo passage of streptolysin-O and albumin across it. [106] As mentioned in the above sections, P. aeroginosa has been detected most frequently in cases of CSOM. In a study done by Eiamprapai et al, using the nested-PCR technique, bacterial DNA of P. aeruginosa and S. aureus were detected in mastoid granulation tissue. [107] Thus the bacteria are obviously able to invade tissue beyond the middle ear. The ability of P. aeruginosa to enter and survive inside the human middle ear epithelial cells (HMEECs) has been demonstrated in a invitro study by Mittal et al.[108] This increases the pathogenicity of the bacteria. But apart from that, this could also translate to a direct toxic effect

on cells, as studies have shown a loss of outer and inner hair cells in the basal turn of the cochlea in COM patients. [102]

With regard to the risk factors for the development of SNHL in COM, the studies are numerous and results are often contradictory.

Khaimanova et al, on a study of 49 patients, presented that the hearing loss was more prominent at higher frequencies and significantly deteriorated with age. There was no influence of duration of disease on the development of SNHL. [109] MacAndie et al, on a study of 41 patients [103] also reported no correlation between duration of disease, or the presence of cholesteatoma or ossicular erosion.

This is in direct disagreement with the study by Kaur et al, where having analyzed the files of 100 patients, report a progressively increasing incidence of SNHL with the duration of the disease. With duration of disease <5 years, the incidence was 13.64% and progressively increased to 33.33% when duration was >26 years. A similar correlation has also been reported by Cusimano.[110] Kasliwal, on studying 1828 patients also report a definite and significant correlation between the duration of disease and SNHL. The SNHL (approximately 27 dB) was found to be significant especially at higher frequencies (2 kHz and 4 kHz).[111] Consistent with the findings reported by Levine [112], they also report a correlation between SNHL and presence of cholesteatoma and erosion of the ossicular chain. However they reported no correlation between the HL and the status of otorrhea.

In another study, Vartiainen et al, studied 874 patients and reported a significant correlation with SNHL and the presence of cholesteatoma and discharge. But they reported no correlation with the type of causative microorganism. In addition this HL was more often found in older patients.[113]

Levine et al, reviewed the charts of 161 patients and stated that there was a significant correlation between SNHL and the presence of cholesteatoma, suggesting that more severe middle ear disease may result in SNHL. Though, on the whole in most of the subjects, the SNHL was not significant.[112] a similar result is quoted by Noordzij et al.[114]

After a review of most of the available literature, it is increasingly apparent that investigations into a possible relationship between COM and SNHL have, time and again, produced a number of conflicting results and conclusions. Chronic Otitis Media. Its occurrence, progression, complications and sequealae is to a large extent dependent on level of nutrition, socio-economic status, cultural practices, availability of medical help and also levels of self-awareness of the disease among the population. Most of the current studies from the Indian subcontinent have been conducted from populations from the Northern parts of India- a region very different from the southern parts of India in terms of culture, social practices, medical health, economy and education. This study aims to extend our current knowledge and understanding on the trends of SNHL in patients of COM among adults (18-60 years) in a South Indian population.

RATIONALE FOR STUDY

RATIONALE FOR STUDY

Our institution, PSG Institute of Medical Science & Research in Coimbatore serves as a referral centre for the southern states of India like Tamil Nadu, Kerala and Andhra Pradesh which predominately run on a large agriculture based economy.

Chronic Otitis Media (COM) is the most common childhood infectious disease worldwide and is the most common cause of hearing impairment in the developing world, although it is infrequently seen in the developed world. It results from an acute ear infection that is not diagnosed promptly or is inadequately treated. The higher prevalence in developing countries is that the cost of treatment is prohibitive. Public education and awareness in developing countries are also an issue. Reviewing the available literature we realized that COM typically occurs in the young child. In this study we explore the association between chronic otitis media and sensorineural hearing loss and correlate between age of patient, duration of disease and the type of COM on the development of sensorineural hearing loss. It is this lack of awareness and need for screening of these individuals at higher risk of developing hearing loss, which prompted us to undertake this study.

AIM OF STUDY

AIM OF STUDY

- To explore the association between chronic otitis media and sensorineural hearing loss.

- To correlate between age of patient, duration of disease and the type of COM on the development of sensorineural hearing loss.

MATERIALS & METHODS

MATERIALS & METHODS

Study design: Cross sectional study.

Study site: Outpatient and Admission cases within the department of ENT, PSG Institute of Medical Sciences, Peelamedu, Coimbatore.

Sample size: 100

Study Period: July 2014 – July 2015

Methods:

- Patients with COM will be selected consecutively as and when they present during the study period based on inclusion and exclusion criteria.
- A detailed history will be taken in all patients and then ENT examination will be carried out to determine the type of COM present. Pure Tone Audiometry will be done to assess for hearing deficit and type of loss.
- The hearing of the patient would be assessed by pure tone audiogram. Hearing loss upto 20 dB will be considered normal, 21-40 dB mild, 40-55 dB moderate, 55-70 dB moderately severe, 70-90 dB severe, and above 90 as profound.
- Categorical data will then be analyzed by **chi-square test**, to correlate the clinical analysis with respect to age of patient, duration of disease and the type of COM on the development of sensorineural hearing loss.

Inclusion Criteria:

History of recurrent otorrhoea occurring within 5 years of the time of data of collection.

Exclusion Criteria:

- Patients in whom hearing loss can be attributed to reasons other than chronic otitis media Eg: Following traumatic perforation, history of long term intake of systemic ototoxic drugs, previous ear surgery, meningitis, enteric fever, head injury, diabetes mellitus, familial hearing loss, labyrinthitis, labyrinthine fistula are to be excluded from the study.
- Patients below the age of 15 years have been excluded due difficulty in obtaining an accurate audiological profile and those above the age of 60 years are not selected so as to exclude the possible effects of aging process.
- Patients not giving consent for the study.

Potential Risks of Study: Nil

Benefits: Data from study may mandate more vigilant detection and management of COM.

National Significance of Study: This study is unlikely to yield results of any national significance.

Data Analysis:

Data collected were entered in Excel Spread sheet and analyzed using STATA statistical software package release 11. I have used the two-sided independent-samples to test and compare means across dichotomous variables (i.e. men v. women); the one-way ANOVA test for comparison of means across categorical data. Qualitative data were given in frequencies with their percentages and quantitative data presented with, Proportions, Mean and standard deviation. Pearson's Correlation analysis was performed to assess the correlation between variables. A type I error of 0.05 was considered in all analyses.

RESULTS

RESULTS

In this study, the results have been evaluated primarily keeping in mind the aim of the study to analyze 100 patients with COM from the outpatient and admission cases within the Department of ENT,PSG Institute of Medical Sciences & Research during the period July 2014 to July 2015 to determine association between chronic otitis media and sensorineural hearing loss and to correlate between age of patient, duration of disease and the type of COM on the development of sensorineural hearing loss.

Detailed evaluation of each case was done comprising of the history, clinical examination including otoscopic examination with otoscope and pure tone audiometry. The clinical data was collected by means of a pro forma and the observations from the audiogram were analyzed with the Master chart as shown in the annexure.

Table 1: Mean Age of the Study Population

	N	Mean	Std. Dev.
Age	100	37.08	11.56

The above table shows that the mean age of the study population is 37 years

Table 2: Gender wise distribution of Age in the Study Population

Variable	N	Mean	Std. Dev.
Women	43	35.86	10.28
Men	57	38.00	12.46
Total	100	37.08	11.56

P Value > 0.05

The above table shows that the male female gender distribution is 57% and 43% respectively. And the mean age of women is 36 years and that of men is 38 years.

Chart 1: Gender wise distribution of Age in the Study Population

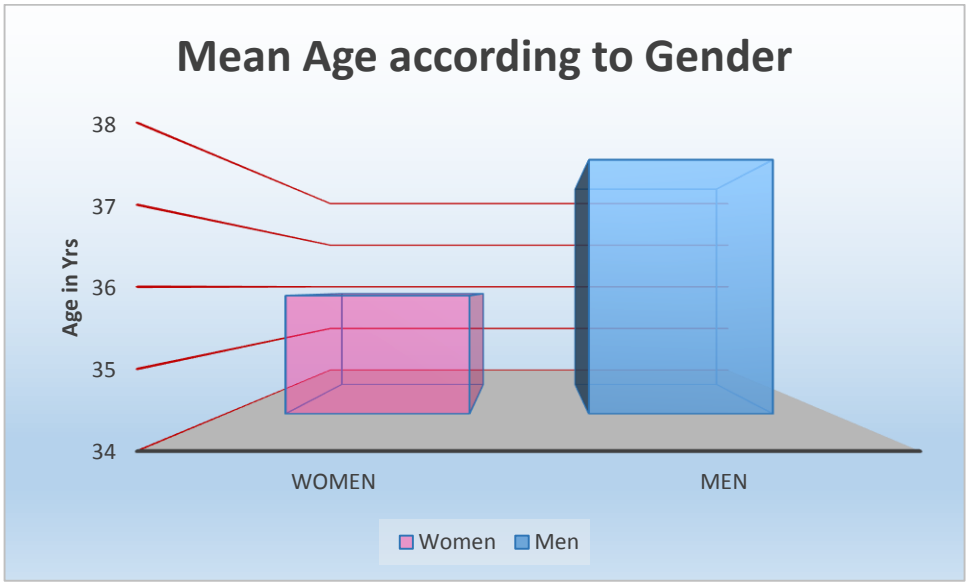


Table 3: Gender wise distribution in the Study Population

Gender	N	Percent
Female	43	43
Male	57	57
Total	100	100

The above table shows that the male female gender distribution is 57% and 43% respectively.

Chart 2: Gender wise distribution in the Study Population

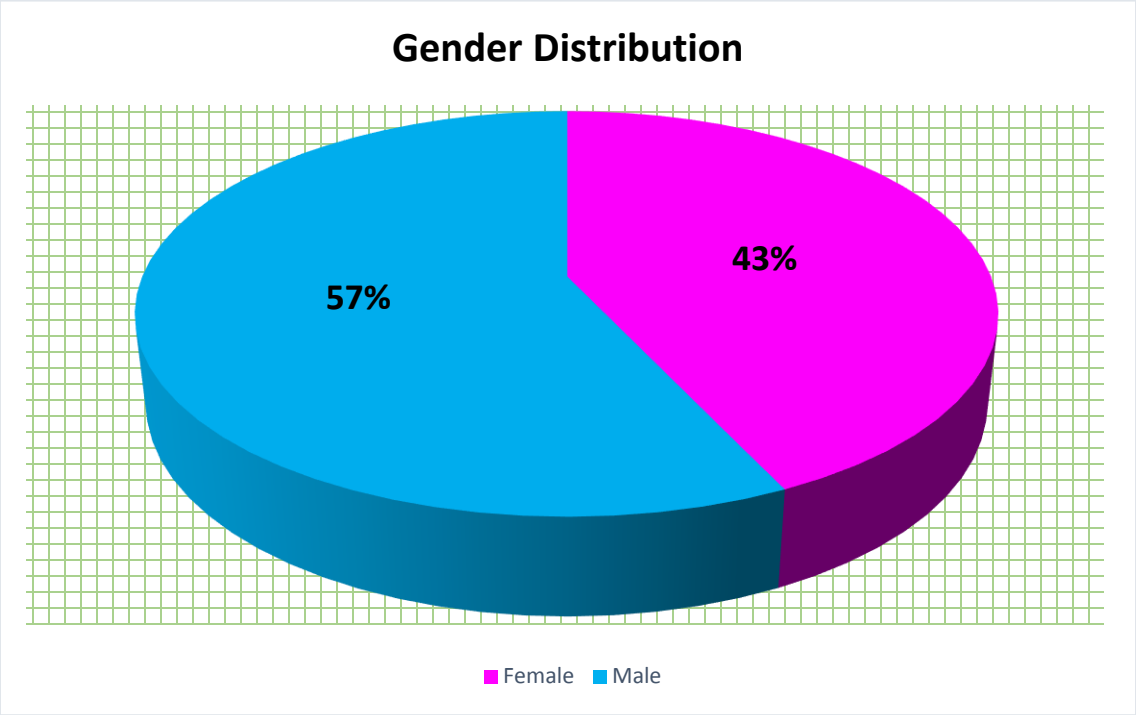


Table 4: Distribution of type of Hearing loss in the Study Population

	Total		Women		Men	
Type	N	Percent	N	Percent	N	Percent
BC	24	24	9	20.93	15	26.32
BM	18	18	7	12.28	11	19.3
LC	5	5	2	4.65	3	5.26
LM	8	8	4	9.3	4	7.02
RC	19	19	8	18.6	11	19.3
RCLM	3	3	2	4.65	1	1.75
RCLS	2	2	0	0	2	3.51
RM	8	8	3	6.98	5	8.77
RMLC	2	2	2	4.65	0	0
RMLS	1	1	0	0	1	1.75
RSLC	4	4	1	2.33	3	5.26
RSLM	6	6	5	11.63	1	1.75
Total	100	100	43	100	57	100

Above table shows the distribution of different types of Hearing loss in the study population.

Key: BC-Bilateral Conductive, BM-Bilateral Mixed, LC-Left Conductive, LM-Left Mixed, RC-Right Conductive, RM-Right Mixed, RCLM-Right Conductive Left Mixed, RCLS Right Conductive Left Sensorineural, RMLC- Right Mixed Left Conductive, RSLC- Right Sensorineural Left Conductive & RSLM - Right Sensorineural Left Mixed

Key: In the previous table the alphabets represents the following,

B – Bilateral

R – Right

L – Left

C – Conductive

S – Sensorineural

M – Mixed

Chart 3: Distribution of type of Hearing loss in the Study Population

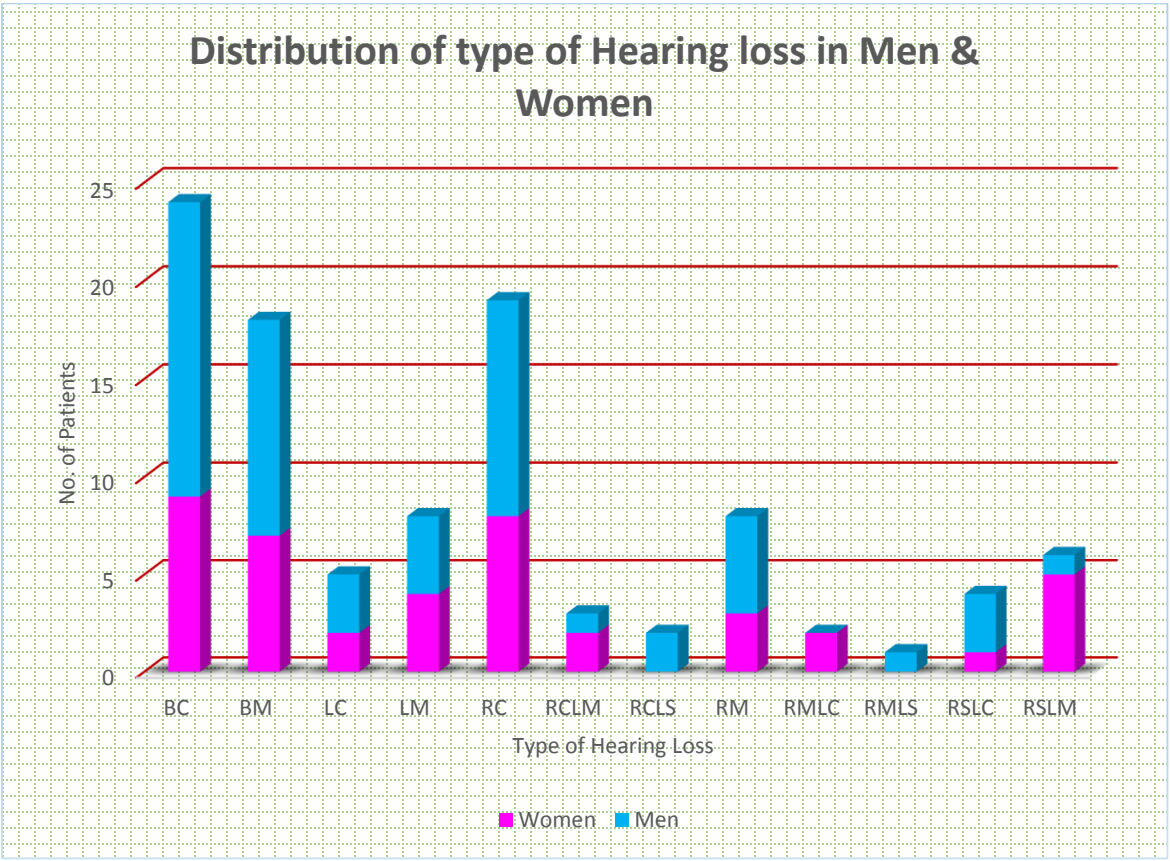


Table 5: Distribution of type of Hearing loss in the Study Population

Type of hearing loss	N	Percent
Conductive	48	48
Others	52	52
Total	100	100

For study purpose, we have classified them as purely "Conductive" variety (Bilateral, Left & Right) and mixed type which encompasses both conductive and sensorineural hearing loss.

The above table shows that the 48% of the study population had conductive type of hearing loss and the remaining 52% had other type of hearing loss.

Chart 4: Distribution of type of Hearing loss in the Study Population

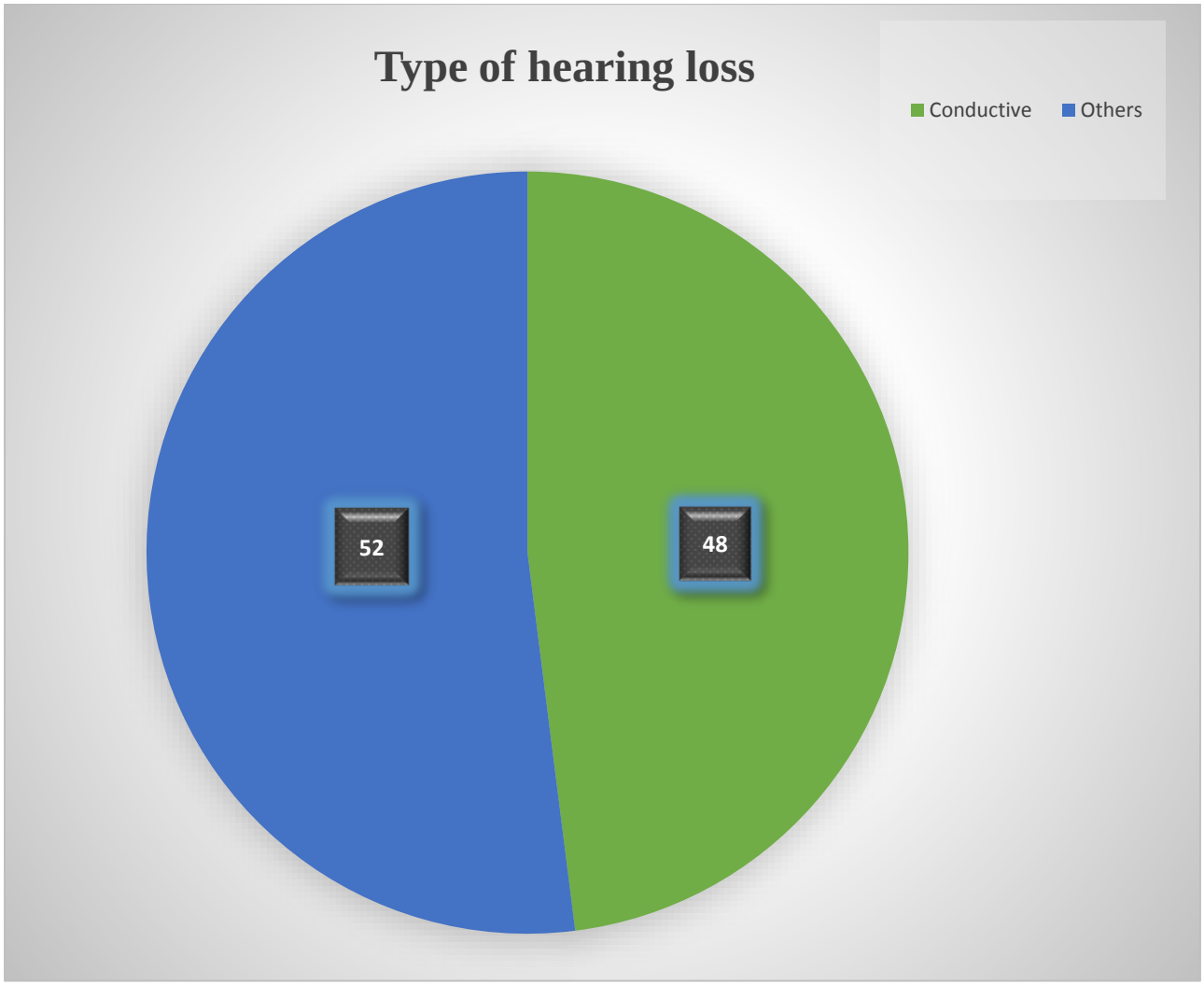


Table 6: Mean Age according of type of Hearing loss in the Study Population

Age	N	Mean	Std. Dev.
Conductive	48	35.40	10.72
Others	52	38.63	12.19

P Value >0.05

The above table shows that the mean age of those with conductive type of hearing loss is 35 years and that of other varieties is 39 years.

The above table shows that there is no statistical difference in mean age between the two categories of hearing loss in the study population.

Chart 5: Mean Age according of type of Hearing loss in the Study Population

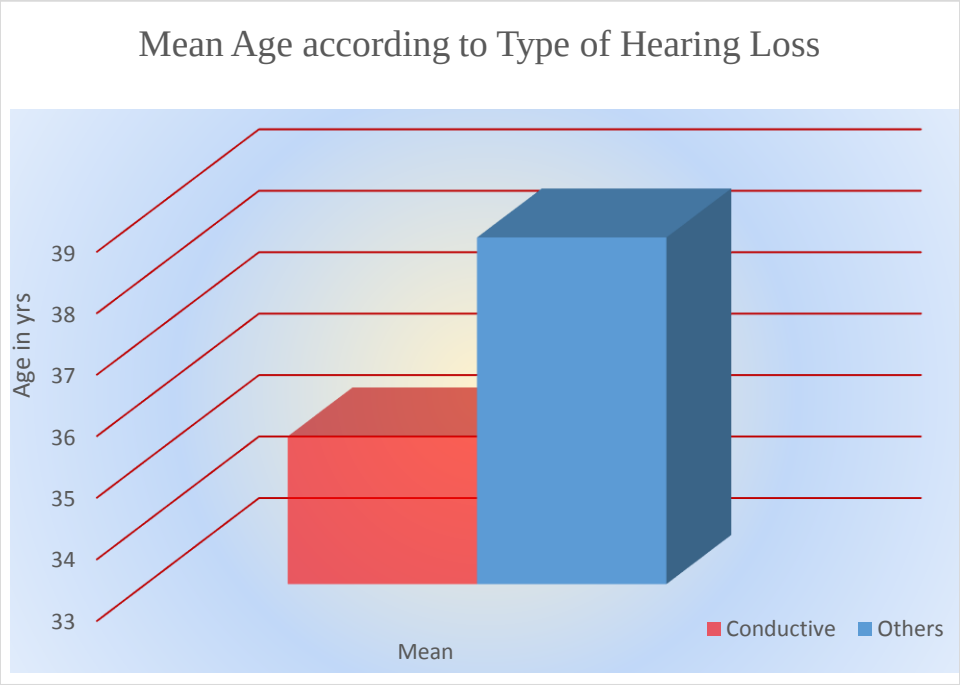


Table 7: Duration of discharge according of type of Hearing loss in the Study Population

Duration of Discharge	N	Mean	Std. Dev.
Conductive	48	6.23	6.92
Others	52	7.61	7.09
Total	100	6.92	

P Value > 0.05

The above table shows that the mean duration of ear discharge of those with conductive type of hearing loss is 6 years and that of other mixed type is 8 years.

The above table shows that there is no statistical difference in mean duration of discharge between the two categories of hearing loss in the study population.

Chart 6: Duration of discharge according of type of Hearing loss in the Study Population

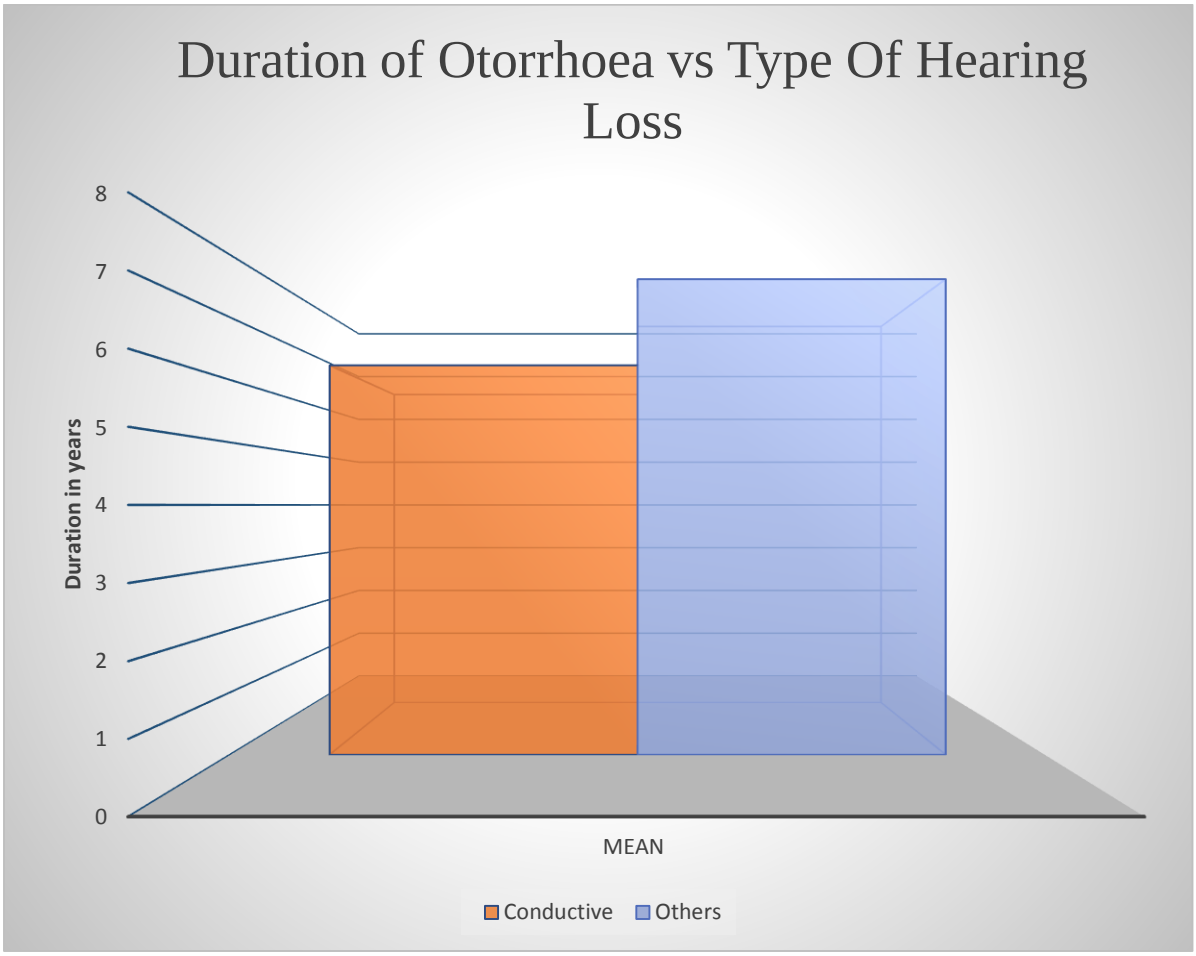


Table 8: Audiogram findings according of type of Hearing loss in the Study Population

	Conductive		Others		P Value
	Mean	SD	Mean	SD	
Right	30.09	12.17	44.37	22.72	<0.01
Left	25.39	10.58	46.81	21.98	<0.01

The above table shows that the mean audiogram findings were 30.09db and 25.39db in right and left ears respectively in those with conductive type of hearing loss. And 44.37db and 46.81db in right and left ears respectively in those with other varieties of hearing loss.

The above table shows that there is a significant statistical difference in mean audiogram values between the two categories of hearing loss in both right and left ear respectively in the study population.

Table 9: Audiogram findings according of type of Hearing loss in the Study Population

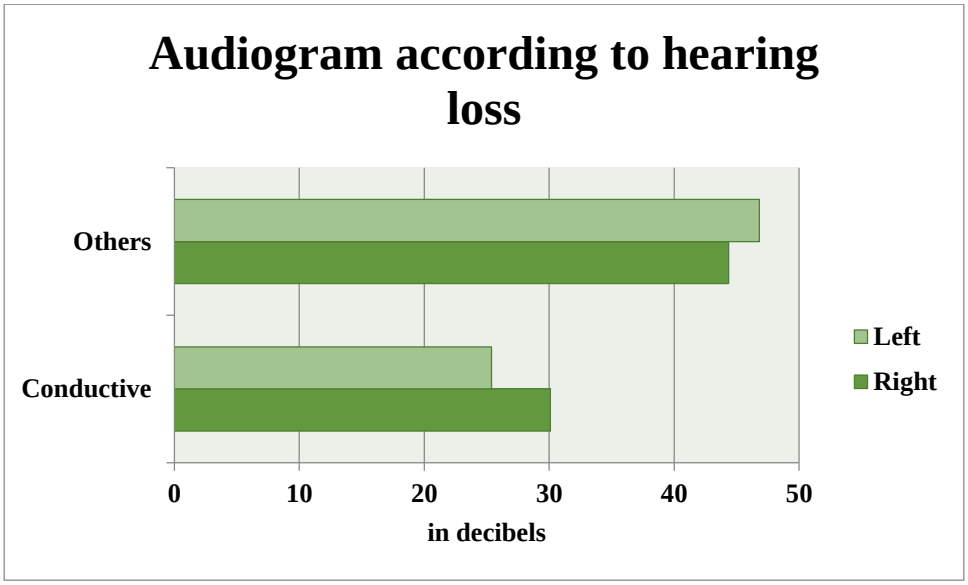


Table 10: Pearson correlation analysis

	Age	Duration of Discharge	Right	Left
Pearson Coefficient	0.1406	0.0986	0.3642	0.5264
P Value	>0.05	>0.05	<0.01	<0.01

Pearson correlation shows as the age, duration of otorrhoea and the audiogram values increases, there are more chances of mixed hearing loss rather than conductive deafness alone

DISCUSSION

DISCUSSION

Chronic Otitis media is one of the commonest ear diseases in developing countries. Tympanic membrane perforation due to COM is one of the common reasons of hearing impairment.

In this study one hundred patients of COM of different age groups were studied. In this 43% were women and 57% were men. This might be due to increased prevalence of COM among male or it might be simple reflection of overall high male attendance in hospital. This might also be mere reluctance of women in our country to come forward for treatment.

48% of them had purely conductive type of hearing loss and the remaining 52% had other types of hearing loss either mixed or sensorineural type.

Among the pattern of hearing loss, this study showed that 48% patients had conductive type of hearing loss, 52% had mixed type of hearing loss. Majority of the studies have proved that conductive type of hearing loss was the most common type of hearing loss following COM[115]. But some studies have demonstrated sensorineural hearing loss occurs in chronic otitis media. One such study showed 7.7% patients suffered from sensorineural hearing loss due to COM[116] & yet another study showed that sensorineural hearing loss occurs particularly in older patients with COM which has usually been present for longer periods[117]. The probable causes are the passage

of toxin through round window membrane that causes biochemical changes in the perilymph and endolymph resulting in gradual destruction of organ of corti.[118]

The mean age of people with conductive hearing loss (35.4 years) was lesser comparative to the mean age in people with other varieties of hearing loss (38.63 years) due to COM.

The mean duration of COM is lesser in patients with conductive type of hearing loss, suggesting that the severity of hearing loss may be associated with increasing duration of disease.[119]

The mean audiogram findings were comparatively higher in the other type of hearing loss compared to the conductive variety in both right and left ear.

Pearson correlation analysis shows as the age of the patient, duration of discharge and the audiogram values increases, there are more chances of sensorineural hearing loss rather than conductive type of hearing loss alone.

LIMITATIONS OF STUDY

LIMITATIONS OF STUDY

The incidence of hearing loss in patients could have been more accurate with a larger group of study population. Only 100 patients with COM were recruited in study due to time and money constraints. The in depth history on the treatment taken, tympanic membrane findings would have enriched the study much more. The association between cholesteatomas and the potential development of SNHL has not been touched upon in the overall analysis of the study.

CONCLUSION

CONCLUSION

From this study it can be concluded that the earlier theoretical knowledge on Chronic Otitis Media causes conductive hearing loss cannot be applied universally. In our study, it is mixed hearing loss which is in higher prevalence than the pure conductive variety in the study population suggestive of the sensorineural contribution. However much more clear idea on type of hearing loss in COM can be documented with further studies involving larger population.

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ANNEXURES

CONSENT FORM

I, **Naveen Zachariah Philip Mathew** am carrying out a study on the topic: "**Analysis of Sensorineural Hearing Loss in Chronic Suppurative Otitis Media"**as part of my research project being carried out under the aegis of the Department of: ENT

My research guide is: Dr. George Zacharias

The justification for this study:

The objectives of this study are:

Primary Objective:

Sample size: 100.

Study volunteers / participants are (specify population group & age group): Patients attending the outpatient services in department of ENT in PSG IMSR between the age group 15 to 60 years.

Location: PSG IMSR.

We request you to kindly cooperate with us in this study. We propose to collect background information and other relevant details related to this study. We will be carrying out:

Initial interview (specify approximate duration): 15 minutes

Data collected will be stored for a period of fifteen years. We will not use the data as part of another study.

Benefits from this study:

Early diagnosis of hearing loss if present, would warrant early management as a means to avert permanent hearing loss.

If you are uncomfortable in answering any of our questions during the course of the interview / biological sample collection, **you have the right to withdraw from the interview / study at anytime.** You have the freedom to withdraw from the study at any point of time. Kindly be

assured that your refusal to participate or withdrawal at any stage, if you so decide, will not result in any form of compromise or discrimination in the services offered nor would it attract any penalty. You will continue to have access to the regular services offered to a patient. You will **NOT** be paid any remuneration for the time you spend with us for this interview / study. The information provided by you will be kept in strict confidence. Under no circumstances shall we reveal the identity of the respondent or their families to anyone. The information that we collect shall be used for approved research purposes only. You will be informed about any significant new findings - including adverse events, if any, whether directly related to you or to other participants of this study, developed during the course of this research which may relate to your willingness to continue participation.

Consent:

The above information regarding the study, has been read by me/ read to me, and has been explained to me by the investigator. Having understood the same, I hereby give my consent to them to interview me. I am affixing my signature / left thumb impression to indicate my consent and willingness to participate in this study (i.e., willingly abide by the project requirements).

Signature / Left thumb impression of the Study Volunteer / Legal Representative:

Signature of the Interviewer with date: Witness:

MASTER CHART

Master Chart

S.No	Age	Sex	Duration of discharge(years)	Right	Left
1	35	Male	2	26.6	16.6
2	40	Female	3	35	18.3
3	38	Male	5	31.6	18.3
4	28	Male	3	19.4	34.5
5	19	Male	5	26.6	16.6
6	36	Male	6	25	26.6
7	29	Male	4	24.6	20
8	26	Male	4	21.6	26.6
9	21	Male	4	58.3	15
10	28	Female	2	40.3	21.5
11	40	Female	2	35.4	18.6
12	49	Male	7	28.2	18.6
13	50	Female	35	20	16.6
14	59	Male	5	26.6	23.3
15	39	Male	5	10	35
16	59	Male	20	21.6	33.3
17	28	Male	2	28.3	21.6
18	26	Male	1	55	13.3
19	46	Male	5	50	40
20	45	Male	20	25	23.3
21	24	Female	2	23.3	51.6
22	46	Male	15	25	23.3
23	29	Male	2	38.3	15
24	41	Male	6	16.6	23.3
25	34	Male	4	20	48.3
26	46	Male	4	25	23.3
27	24	Male	3	32.4	14.2
28	36	Female	4	26.6	16.6
29	41	Female	10	35.4	20.7
30	31	Female	4	21.6	21.6
31	16	Male	4	35	33.3
32	26	Female	9	25	18.3
33	47	Male	5	60	23.3
34	55	Male	5	25	20
35	35	Female	1	26.6	46.6
36	33	Female	2	28.5	15
37	56	Male	25	56.3	33.3
38	30	Female	5	20	21.6
39	40	Female	20	28.3	25

S.No	Age	sex	Duration of discharge	right	left
40	36	Female	9	50	20
41	37	Male	5	15	21.6
42	22	Female	2	23.3	58.3
43	28	Male	1	46.6	36.6
44	32	Male	2	23.3	21.6
45	30	Female	1	16.6	21.6
46	40	Female	2	23.3	21.6
47	21	Female	2	50	20.3
48	22	Female	5	18.3	45
49	22	Male	1	35	16.6
50	30	Female	3	55	26.6
51	42	Male	1	65	76.6
52	20	Male	2	93.3	31.6
53	57	Male	4	31.6	25
54	36	Female	5	42.8	14.2
55	48	Male	6	90	20
56	21	Male	20	65	85
57	49	Male	3	73.3	63.3
58	19	Female	5	41.6	51.3
59	39	Female	8	73.3	90
60	57	Female	20	50	71.6
61	32	Male	3	21.6	65
62	25	Male	1	43.3	45
63	47	Female	1	16.6	41.6
64	46	Female	6	38.3	30
65	48	Male	0.5	26.6	21.6
66	32	Male	4	38.3	40
67	41	Female	10	23.3	38.3
68	54	Male	17	24.6	58.2
69	26	Male	5	53.3	30
70	50	Female	20	36.6	50
71	34	Female	9	16.6	33.3
72	33	Female	4	23.3	68.3
73	45	Male	3	33.3	21.6
74	25	Male	10	21.6	65
75	40	Male	20	53.3	40
76	37	Female	10	18.3	48.3
77	40	Female	6	51.6	20
78	34	Female	7	23.3	63.3
79	24	Female	2	16.6	60

S.No	Age	sex	Duration of discharge	Right	Left
80	18	Male	1	28.3	93.3
81	50	Male	17	60	21.6
82	46	Female	15	66.6	40
83	47	Male	12	81.6	60
84	46	Female	6	33.3	16.6
85	32	Male	3	73.3	90
86	48	Male	10	30	70
87	46	Male	3	31.6	33.3
88	23	Female	5	63.3	68.3
89	48	Female	1	103.3	45
90	40	Female	6	95	30
91	53	Female	7	30	70
92	57	Male	10	36.6	36.6
93	19	Male	4	16.6	33.3
94	19	Female	5	36.6	58.3
95	51	Male	23	48.3	20
96	41	Male	8	21.6	35
97	55	Female	5	55	85
98	59	Male	35	36.6	35
99	20	Female	1	40	28.3
100	38	Male	2	23.3	53.3